EXHIBIT 11

REPORT FOR ZOLOFT FEDERAL LITIGATION

Expert Report of Stephen E. Kimmel, MD, MSCE, FACC, FAHA, FISPE

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I am submitting this report in connection with litigation regarding whether Zoloft (sertraline), an antidepressant medication manufactured by Pfizer, can cause congenital heart defects.

I. Introduction

I am a Professor of Medicine with tenure in the Department of Medicine at the University of Pennsylvania (Penn), a Professor of Epidemiology in the Department of Biostatistics and Epidemiology at Penn, a Senior Scholar in the Center for Clinical Epidemiology and Biostatistics (CCEB), and a Senior Fellow of the Leonard Davis Institute at Penn. I am also the Director of the Clinical Epidemiology Unit (CEU) of the CCEB and of the Epidemiology Division of the Department of Biostatistics and Epidemiology, and Director of the Center for Therapeutic Effectiveness Research at Penn.

Most of my professional career has been devoted to studying, teaching, and writing on the science of pharmacoepidemiology – the recognized scientific methods by which one can assess whether or not a drug is associated with or can cause or prevent disease. I have authored and edited reference books and textbooks (including perhaps the most comprehensive book in the field), book chapters, and numerous peer-reviewed articles concerning pharmaco-epidemiology, and have lectured widely on the subject. I am also a board certified cardiologist.

I previously submitted a detailed expert report addressing the issue of Zoloft (sertraline) and birth defects – specifically including congenital heart defects – in the federal MDL litigation involving Zoloft, *In re Zoloft (Sertraline Hydrochloride) Products Liability Litigation*, MDL No. 2342 (E.D. Pa.) (Rufe, J.). A copy of my MDL report, dated September 12, 2013, which also includes a description of my qualifications, a copy of my CV, and a list of references, is attached

as Ex. A, and is incorporated by reference. As I explain in my MDL report, it is generally accepted that:

> "Numerous studies have been conducted to investigate a possible association between SSRIs and birth defects, and substantial data confirm that there is no evidence of a causal link between Zoloft and any birth defects. Moreover, expert regulatory authorities in the United States and other countries have independently assessed the data and have publicly declared that a causal link has not been established, as have independent professional scientific associations."

Ex. A at 4.

This updated report is divided into two sections:

In Section I, I discuss several recent studies (published after my prior report) which confirm the lack of an association between Zoloft and cardiac defects. In fact, these newer studies address many of the methodological limitations of prior studies, including power, the ability to account for confounding, and the possibility of other biases. The most recent study (Huybrechts 2014) – which addressed these concerns – demonstrated no association between Zoloft and cardiac defects. In addition, several professional organizations, including the American Heart Association, have confirmed and continue to confirm, the previously generally accepted position that there is no increased risk of congenital heart defects from Zoloft. Details of these new studies and reports are provided in Section I, below.

In Section II, I discuss the opinions of a new expert, statistician Nicholas Jewell, PhD, who has filed a report concerning Zoloft and cardiac defects. Dr. Jewell was retained by plaintiffs subsequent to my prior report, in lieu of plaintiffs' prior expert, Dr. Anick Bérard. Dr. Jewell's report contains numerous methodological flaws, inaccuracies, and inconsistencies. Dr. Jewell's methodological errors include the following:

1. Use of non-independent data sources to improperly claim replication of certain study findings, and use of data from one type of cardiac defect to improperly claim consistency with findings for other defects.
2. Selective reliance on only certain portions of data to examine confounding, and improper dismissal or misrepresentation of objective evidence demonstrating that confounding is a key source of false positive findings in earlier studies.
3. Misapplication of established epidemiological methods to improperly dismiss findings that contradict Dr. Jewell's opinion.
4. Use of inconsistent and different standards depending on whether the findings being considered support his conclusion compared with results that refute his conclusion.

For the reasons set forth in my original MDL report and this report, it is my opinion that there is no evidence of a causal association between Zoloft and congenital heart defects.

II. New Studies and Articles Confirm the Lack of Evidence of Causation

Based on the literature that I reviewed in my previous expert report and the new literature that has since become available, there are several conclusions that one must draw: First, there has been a lack of independent replication of a Zoloft-cardiac defect association. Second, confounding is clearly an important factor in explaining an apparent (false) relationship between Zoloft and cardiac defects. Third, the inclusion of only live births in epidemiological studies is unlikely to have biased the results in a manner that would have masked a true association between Zoloft and cardiac defects if one existed. Fourth, misclassification of exposure is similarly unlikely to have masked an association and, in several cases, would likely create a false association.

In the discussion below, I will review both the original research and summaries of evidence that have been published since my prior report.

A. Huybrechts (2014)

Huybrechts et al. recently published their study in the *New England Journal of Medicine*. This study is an important addition to the scientific literature because it was able to address several potential limitations of prior studies, including confounding, power, and bias.

Specifically, the authors of all prior studies in the literature have raised concerns that confounding by indication created false associations between Zoloft and birth defects in their studies. Huybrechts et al. addressed these concerns to a much greater degree than prior studies and clearly and objectively demonstrated the importance of confounding in creating false positive associations. Similarly, concerns have been raised about study power (the ability to detect an association assuming one truly exists) because of prior studies' sample sizes. Huybrechts et al. was orders of magnitude larger than all prior studies and was able to address the concerns about study power. With respect to bias, Huybrechts et al. performed numerous analyses to address these concerns and determined that potential biases would not meaningfully mask a true association between Zoloft and cardiac defects if one indeed existed.

1. Well Powered to Detect an Association, if One Truly Existed

This was the largest cohort study to date, including almost 1 million women and 14,040 women exposed to Zoloft. This study population is orders of magnitude larger than prior studies, including the Danish cohort studies (the only cohort that demonstrated an association between Zoloft and cardiac defects as a group): for example, with respect to the number of women exposed to Zoloft, Huybrechts et al. was 54-times larger than Pedersen et al., 40-times larger than Kornum et al, and 17-times larger than Jimenez-Solem et al.

In fact, Huybrechts included as many cardiac defects (129) as all other non-overlapping studies cited by Dr. Jewell (in his report) combined (Jimenez-Solem, Louik, Colvin, Alwan, Reis-Kallen 2010, Malm, and Ban – a total of 130). The study had sufficient power to identify an association between Zoloft and cardiac defects if one existed. If one uses the event rate in the unexposed population, with 11,056 women exposed to Zoloft in the depression-restricted cohort, the study has essentially 100% power to detect a relative risk of 2 for cardiac defect. That is, the study had sufficient size to rule out a 2-fold increase in risk from Zoloft. Similar calculations for the specific cardiac defects examined in the study, ventricular septal defects, right ventricular outflow track obstructions, and other cardiac malformations, reveal substantial power to identify a two-fold increased risk if one truly existed: 100%, 90%, and 100%. Notably, the study did not demonstrate an association with any of these defects.

The study used a nationwide Medicaid dataset and demonstrated that Zoloft is not associated with cardiac malformations overall (odds ratio (OR) of 1.09 and 95% confidence interval (CI) of 0.88–1.34). It is important to note that, with such a large study population, the study demonstrated a precise OR of one, and clearly ruled out a relative risk of 2 or more. Analyses of specific groups of cardiac malformations revealed similar findings: ventricular septal defects (VSD, OR 1.04; 95% CI: 0.76–1.41), right ventricular outflow tract obstruction (RVOT, OR 1.12, 95% CI: 0.67–1.88), and other cardiac defects (OR 1.19, 95% CI: 0.89–1.59). These data clearly do not demonstrate an association between Zoloft and cardiac defects and do not replicate prior associations reported in the literature.

2. Strong Confirmation of Confounding by Indication

As noted above and in my prior report, all scientists who have studied SSRIs and birth defects have been concerned about the effects of confounding by indication: that Zoloft is given to women with depression and that, because depression itself alters behaviors and risk factors in

many ways that could increase the risk of birth defects, the apparent higher risks in comparisons of women who needed Zoloft compared with women who did not need antidepressants could be due to the underlying condition during pregnancy rather than the drug.

My prior report details how these differences have been documented in prior studies. Huybrechts et al. provides even further evidence for these important differences. As noted in their Supplementary Appendix table S11, women who were prescribed Zoloft were markedly different from women unexposed to antidepressants. For example, compared with women who did not receive antidepressants during pregnancy, women prescribed Zoloft were more likely to have other mental and physical health disorders: 4.7 times more likely to have other mental and physical health disorders, 3.2 times more likely to have pain related diagnoses, 8.5 times more likely to have sleep disorders, 2.3 times more likely to have diabetes prior to pregnancy and 1.6 times more likely to have gestational diabetes, and 3.2 times more likely to have epilepsy, to name only a few differences. They were also older, 2.7 times more likely to smoke, substantially more like to use other psychotropic medications (4 times more likely to use anticonvulsants, 3.5 times antipsychotics, 6.8 times anxiolytics, 5.2 times benzodiazepines, 2.6 times barbiturates, and 2.2 times other hypnotic drugs), and 2.6 times more likely to use suspected teratogens.

The critically important point here is that women who need Zoloft during pregnancy are markedly different than women who do not need antidepressants. This is exactly the concern that authors have had in prior studies.

Huybrechts et al. was better able than all prior studies to address these differences. The primary approach was to both limit the study population to those women with a diagnosis of depression between their last menstrual period through the end of the first trimester (of note, this is the exact methodology that McDonagh et al., discussed below, call for in their recent meta-

analysis) and to further adjust for confounders (such as comorbidities, other medication use, age, parity, multiple gestation, other mental health disorders, etc) using a propensity score method.

It is critically important to note that just restricting the cohort to depressed women did not sufficiently account for the differences between Zoloft users and non-users. The authors state: "substantial differences in the characteristics of the patients remained after the cohort was restricted to women with depression. Stratification according to the propensity score ensured that comparisons were made between groups with nearly identical characteristics." The change from the unadjusted odds ratio after adjustment for confounding was a decrease of 14%, providing substantial and objective evidence of the importance of accounting for confounding. This same reduction in the OR with adjustment was demonstrated for the specific cardiac defects examined. The authors did even further analyses to account for confounding (the high-dimensional propensity score adjustment) and the OR for Zoloft and all cardiac malformations was further reduced (to 1.06, 95% CI: 0.86-1.32).

In short, this study clearly demonstrates the importance of accounting for confounding when assessing the relationship between Zoloft and cardiac defects.

3. Findings are not Attributable to Bias

With respect to misclassification of exposure (i.e., filling a prescription does not necessarily mean that a woman took the drug), the study performed analyses to determine if misclassification of exposure to SSRIs could bias the results. They examined dispensing of at least two prescriptions of an SSRI during the first trimester and the results were unchanged: OR for cardiac defects using one or more dispensing of Zoloft 1.19 (95% CI: 0.95, 1.49) versus the OR using two or more dispensing of Zoloft of 1.17 (95% CI: 0.82-1.67). These results demonstrate that misclassification of exposure from the use of prescription databases is unlikely to bias the study results. (It is also important to note that these results are much more precise

than those of Pedersen, which was a much smaller study.) Of note, they also performed a dose-response analysis and reported no dose-response relationship.

With respect to ascertainment bias (i.e., that birth defects were missed or misclassified), the authors performed several analyses. First, they included any cardiac diagnoses made up to a year after birth and still found no relationship between Zoloft and cardiac defects. Second, they performed an analysis to determine the potential bias that could be due to misclassification of cardiac malformations and again found no association (OR for Zoloft of 1.12, 95% CI: 0.86-1.48). Third, they confirmed previous associations of other exposures that have well-known associations with cardiac defects.

Finally, the authors performed analyses to determine if the exclusion of spontaneous abortions, stillbirths, or pregnancy terminations for cardiac anomalies from the study could bias the results. They conclude that "differences in the proportion of terminations among women with depression on SSRIs vs. those untreated within levels of covariates used in the adjustment would have to be unrealistically strong in order to dilute a relative risk of 1.5 or higher. Moreover, this selection bias would not explain the discrepancies with prior studies, since they also included live births preferentially."

4. Study Limitations do not Meaningfully Affect the Results

Like all studies, Huybrechts et al. has several limitations. It only included live births (although, as noted above, their analyses did not suggest that this was a meaningful bias). They relied on ICD-9 codes for confounding (which could leave residual, unaccounted for, confounding), and for identifying malformations, although they used a validated algorithm to identify these defects (Margulis AV, Setoguchi S, Mittleman MA, Glynn RJ, Dormuth CR, Hernández-Díaz S. Algorithms to estimate the beginning of pregnancy in administrative databases. Pharmacoepidemiol Drug Saf 2013; 22:16-24.)).

Like almost all studies they had to estimate gestational age, and they did not include the outcomes from ICD code 745.5. This latter is the ICD code for several diagnoses including ostium secundum atrial septal defect (ASD) and patent foramen ovale. The former, ASD can be mistaken for the latter (PFO), and PFO is not considered a major cardiac birth defect. The authors note that their aim was "to exclude conditions that are generally considered physiologically normal; i.e., patent foramen ovale (PFO)" and that ICD codes "do not allow us to differentiate PFO - the most common type - from other types of ASD." Further, they "wanted to exclude technology-induced defects; i.e., minor defects with little clinical significance that are diagnosed only because of intense testing with sensitive technology, such as very small ASDs." They state that "Inclusion of a large number of PFOs which are not real malformations would likely have biased the results towards the null for cardiac malformations overall (that is, inclusion of ASDs would have resulted in odds ratios closer to 1)."[1] I agree with their assessment. Further, they do include ICD codes for other ASDs, including ostium primum and sinus venosus defects. Thus, the exclusion of code 745.5 would not limit the ability to identify associations with these defects nor would it affect their analyses of VSDs or RVOT defects.

5. Conclusion: Zoloft is not Linked to Congenital Heart Defects

Thus, Huybrechts et al. provide important new information that: confirms the lack of association between Zoloft and cardiac defects; demonstrates the importance of confounding in explaining apparent associations between Zoloft and cardiac defects; does not support the hypothesis that misclassification of exposure to SSRIs in prescription database studies meaningfully biases study results; and illustrates that ascertainment bias from either the use of

[1] Krista Huybrechts email dated February 27, 2015. See also National Birth Defects Prevention Network, Guidelines for Conducting Birth Defects Surveillance June 2004, at A3.2-20; EUROCAT, Guide 1.4, Chapter 3.2, Minor Anomalies for Exclusion, 2013 (Oct. 14, 2014).

ICD codes or the exclusion of pregnancy terminations is highly unlikely to mask a true association between SSRIs and cardiac defects. Thus, overall, confounding is a major concern in observational studies and one must consider a study's ability to account for confounding before one takes the study results at face value. (NB, one must still also consider other sources of study error as well, such as recall bias, multiple comparisons, etc.)

B. Ban (2014)

Ban et al. published a cohort study from the UK that attempted to account for depression and other confounders, albeit not as thoroughly as Huybrechts et al. The study did not identify a statistically significant relationship between Zoloft and cardiac defects.

In Ban, the primary comparison group was women without a clinical diagnosis of depression, but this was only "so that our results were comparable with the published literature [that did not do comparisons among only women with depression], so that the size of the risk estimates for diagnosed unmedicated depression could be directly compared with those for antidepressant drug exposures, and to maximise the statistical power." This should not imply that this comparison group is the optimal one, and the authors thus did do comparisons with women with depression not exposed to antidepressants, a more relevant comparison, as noted by McDonagh, et al.

Their primary result for Zoloft and cardiac defects was an OR of 1.52 (95% CI: 0.78-2.96) when compared with women without depression. However, when compared with women with depression, the more appropriate comparison group, the OR for Zoloft was 1.39 (95% CI: 0.70-2.74, P=0.345), a reduction in the OR of 9% and consistent with confounding. The study limitations included limited adjustment for confounding; the potential for chance findings; limited power in sub-group analyses; exclusion of abortions, stillbirths, or pregnancy terminations for cardiac anomalies; and inability to confirm actual use of SSRIs. With respect to

adjustment for confounding, the study relied on a diagnosis of depression any time within a year prior to conception; therefore, it did not actually measure depression at the time of pregnancy, thus diminishing the ability to account for depression in the analyses.

It is important to further discuss the point made above. The concerns about confounding by depression relate to the fact that active depression alters behaviors and risk factors in women who have depression during pregnancy in many ways that could increase the risk of birth defects. However, depression, unlike for example Type 1 diabetes, waxes and wanes. Many patients with depression will respond to treatment and no longer have depression symptoms after several months of treatment.[2] Thus, having a diagnosis of depression months before pregnancy does not mean that a patient has depression during pregnancy. In one study, it was suggested that 46% of women with a depression diagnosis 39 weeks before pregnancy no longer had depression during pregnancy. (Dietz PM, Williams SB, Callaghan WM, Bachman DJ, Whitlock EP, Hornbrook MC. Clinically identified maternal depression before, during, and after pregnancies ending in live births. *Am J Psychiatry*. 2007 Oct;164(10):1515-20.)

Ban's definition of depression included any diagnosis of depression in the year before conception; thus it is highly likely that many women in their unmedicated-depressed comparison group did not actually have depression during pregnancy. This is supported by Huybrechts et al where, even within the depression-restricted cohort, there were still differences in the clinical characteristics of women who used antidepressants during pregnancy compared with those who

[2] See, for example: Papakostas GI, Fava M. Does the probability of receiving placebo influence clinical trial outcome? A meta-regression of double-blind, randomized clinical trials in MDD. *Eur Neuropsychopharmacol*. 2009 Jan;19(1):34-40. Gartlehner G, Hansen RA, Morgan LC, Thaler K, Lux L, Van Noord M, Mager U, Thieda P, Gaynes BN, Wilkins T, Strobelberger M, Lloyd S, Reichenpfader U, Lohr KN. Comparative benefits and harms of second-generation antidepressants for treating major depressive disorder: an updated meta-analysis. *Ann Intern Med.* 2011 Dec 6;155(11):772-85.

did not: Women still using antidepressants during pregnancy are likely to still have depression while those who had depression in the past but are not using antidepressants are likely to no longer have depression.

Further, also in contrast to Huybrechts et al, Ban did not adjust for many potentially confounding variables (e.g., anticonvulsant medications, other indications for antidepressants, multifetal pregnancy, etc). The authors also note that stillbirths were unlikely to account for their findings, although they do not do analyses to determine the potential effects of only including live births. The authors also do not adjust for multiple comparisons but do state that they would expect "smaller values of $P < 0.01$ to be less likely as a result of chance alone." As noted above, the P-value for Zoloft was 0.345.

In sum, Ban confirms the absence of a significant association between Zoloft and congenital heart defects, and provides additional evidence regarding the importance of confounding.

C. McDonagh (2014a, 2014b)

McDonagh et al. published both a peer-reviewed paper and a peer-reviewed full report (Evidence Report/Technology Assessment) that were both a systematic review and meta-analysis of depression drug treatment outcomes in pregnancy and postpartum. The importance of these papers is that the authors use a pre-defined, objective approach to summarizing the data and to determining, *a priori*, which studies to include in specific analyses (i.e., they used a systematic method to guide this analyses rather than one that might be biased by knowing the individual study results in advance).

Because of the underlying concern of confounding, the authors determined that only studies examining the effects of antidepressants among women with an indication for antidepressant use (either an underlying diagnosis or the use of a different antidepressant drug)

provided "direct evidence." Studies comparing women receiving antidepressants for any reason with women receiving no antidepressant and unknown depression status were considered "indirect evidence." The importance of this distinction is the authors' acknowledgement of the need to account for the underlying indication in studies of SSRIs and birth defects.

It is also important to note that neither the Huybrechts nor the Ban papers were available to the McDonagh et al. during their selection of studies for inclusion.

McDonagh et al. rated the risk of bias based on predefined criteria established by the Drug Effectiveness Review Project.[3] All studies were rated by one reviewer and checked by another reviewer. All disagreements were resolved through consensus.

There were insufficient data from "direct evidence" studies to draw conclusions about Zoloft and congenital malformations (as noted above, McDonagh did not have the Huybrechts or Ban studies, both providing "direct evidence," available for their analysis). There were nine "indirect evidence," medium and low risk of bias studies examining Zoloft. Based on the 7 of these studies that reported adjusted ORs, the OR for Zoloft and major malformations was 0.98 (95% CI: 0.85-1.13). When they performed their pre-specified sensitivity analysis, thereby removing studies that did not adjust for at least three of the four key confounding factors identified for their analyses, they were left with four studies and "a more precise estimate (pooled adjusted OR 0.92; 95% CI: 0.80 to 1.05)."

When examining cardiac malformations, the OR for Zoloft, based on 6 medium to low risk of bias studies that reported adjusted ORs, was 1.08 (0.70 to 1.65). Because of statistical

[3] Based on the adequacy of the patient selection process, whether important differential loss to followup or overall high loss to followup occurred, the adequacy of exposure and event ascertainment, whether acceptable statistical techniques were used to minimize potential confounding factors, and whether the duration of followup was reasonable to capture investigated events.

heterogeneity (i.e., variability in the odds ratios), the authors also performed a sensitivity analysis whereby they removed studies that that did not adjust for at least three of the four *a priori* identified potential confounding factors, resulting in a pooled OR of 0.76 (95% CI: 0.59-0.97). Further limiting the analysis to studies that also indicated efforts to identify serious cardiac malformations yielded a pooled estimate of 0.76 (95% CI: 0.57 to 1.00; reported p-value of 0.51) and eliminated all statistical heterogeneity. (Of note, the P-value reported for this last analysis appears to be a typo and should be 0.05, based on the confidence interval.) It is important to note, with respect to power, that this meta-analysis revealed a 95% confidence that essentially excluded an increased risk from Zoloft on cardiac malformations.

Although the authors used rigorous methods for study identification, criteria of quality, and data analyses, the study is somewhat limited by the inclusion of overlapping data in one of their analyses. Specifically, two Danish studies (Kornum and Pedersen) and two Swedish studies (Kallen and Reis) with overlapping populations were included in the overall OR for cardiac defects. However, in the analyses that excluded "studies that did not adjust for at least three of the four potential confounding factors identified for this review" and those that "indicated efforts to identify serious cardiac malformations," this overlap was apparently eliminated and, as noted above, the resulting OR was 0.76 (95% CI: 0.57 to 1.00).

D. Bérard (2015)

A paper by Bérard et al. was recently published as an unedited manuscript in the journal *American Journal of Obstetrics and Gynecology*.[4] This was a study of 18,493 pregnancies from Quebec, 1998-2010. The paper examined the relationship between antidepressants (separating

[4] The publication states "The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain."

out Zoloft specifically) and multiple birth defects. The study examined women with a diagnosis of depression and/or anxiety or exposed to antidepressants in the 12 months before pregnancy[5] and performed multiple comparisons between antidepressant drugs (Zoloft, non-Zoloft SSRIs, and non-SSRI antidepressants) and those unexposed to antidepressants. Table 2 lists 33 different comparisons. Multivariable adjustment was made only for maternal age, welfare status, diabetes, hypertension, asthma, and other medication use. The study did not find a statistically significant relationship between Zoloft and cardiac malformations (OR 1.16, 95% CI: 0.62-2.19). It did however identify two statistically significant relationships with Zoloft (ventricular/septal defects and craniosynostosis).

There are several limitations to the study, including multiple comparisons and insufficient adjustment for confounders. However, even apart from those limitations, there is an overarching, major concern, that makes the results totally unreliable at this time. The confidence intervals (and thus the statistical significance) for the only two statistically significant findings are not consistent with any of the other analyses, nor with what would be expected from the data. Thus, specific to septal defects, despite the fact that there are fewer events relative to the analysis for all cardiac malformation combined, the confidence interval for septal defects is narrower for the septal defects analysis (not wider as one would expect). I have computed crude (unadjusted) ORs and CIs for all results in Table 2 and can confirm all results for Zoloft except for the two that are stated to be statistically significant.[6] The results are shown in the table below:

[5] Similar to Ban et al., this means that many women were likely to not actually be depressed during pregnancy in the comparison (unexposed) group.

[6] Because the tests I performed did not use the same model that Berard et al. used, the results differ slightly, usually only at the 2nd decimal point. However, the results are essentially the same except for the 2 exceptions noted.

Bérard (2015) Possible Miscalculations

1st Trimester Sertraline Exposure

	Congenital Malformation	Odds Ratio	Confidence Interval
Bérard (2015)[7]	Major congenital	1.13	(0.83-1.55)
openepi.com[8]	Major congenital	1.12	(0.82-1.54)
Bérard (2015)	Nervous System	1.78	(0.73-4.34)
openepi.com	Nervous System	1.75	(0.71-4.3)
Bérard (2015)	Eye, ear, face and neck	0.47	(0.06-3.39)
openepi.com	Eye, ear, face and neck	0.47	(0.06-3.39)
Bérard (2015)	Circulatory system	1.31	(0.75-2.30)
openepi.com	Circulatory system	1.31	(0.74-2.29)
Bérard (2015)	Respiratory system	N/A	N/A
openepi.com	Respiratory system	N/A	N/A
Bérard (2015)	Digestive system	1.15	(0.47-2.83)
openepi.com	Digestive system	1.16	(0.47-2.83)
Bérard (2015)	Genital organs	0.98	(0.35-2.72)
openepi.com	Genital organs	1.02	(0.37-2.75)
Bérard (2015)	Urinary system	0.86	(0.27-2.72)
openepi.com	Urinary system	0.86	(0.27-2.72)
Bérard (2015)	Musculoskeletal system	1.06	(0.64-1.75)
openepi.com	Musculoskeletal system	1.05	(0.63-1.75)
Bérard (2015)	Cardiac malformations	1.19	(0.63-2.25)
openepi.com	Cardiac malformations	1.19	(0.62-2.24)
Bérard (2015)	Ventricular/atrial septal defect	1.35	(1.01-1.79)
openepi.com	Ventricular/atrial septal defect	1.35	(0.69-2.65)
Bérard (2015)	Omphalocele	N/A	N/A
openepi.com	Omphalocele	N/A	N/A
Bérard (2015)	Craniosynostosis	1.94	(0.99-3.81)
openepi.com	Craniosynostosis	1.94	(0.61-6.21)
Bérard (2015)	Cleft palate	N/A	N/A
openepi.com	Cleft palate	N/A	N/A

Based on these calculations, there is no statistically significant association between Zoloft and either septal defects or craniosynostosis. Until these discrepancies can be resolved, the findings of this paper must be deemed unreliable.

[7] Bérard, et al, Sertraline Use During Pregnancy and the Risk of Major Malformations, *American Journal of Obstetrics and Gynecology* (2015), doi: 10.1016/j.ajog.2015.01.034.

[8] http://openepi.com/TwobyTwo/TwobyTwo.htm

E. Donofrio (2014)

In April 2014, the American Heart Association released a document, "Diagnosis and Treatment of Fetal Cardiac Disease: A Scientific Statement From the American Heart Association." An independent group "reviewed the available literature pertaining to topics relevant to fetal cardiac medicine, including the diagnosis of congenital heart disease and arrhythmias, assessment of cardiac function and the cardiovascular system, and available treatment options." In addition, the "American College of Cardiology/AHA classification of recommendations (COR) and level of evidence (LOE) were assigned to each recommendation according to the 2009 methodology manual for American College of Cardiology/AHA Guidelines Writing Committee."

The review concludes that "there is no increased risk of CHD [congenital heart disease] associated with the use of most SSRIs, although paroxetine may be an exception." Their recommendations are that "Referral for fetal cardiac evaluation is not indicated for …. maternal medications including SSRIs (other than paroxetine) (Class III; Level of Evidence A)."[9]

The AHA Scientific Statement was approved by the American Heart Association Science Advisory and Coordinating Committee and endorsed by both the American Society of Echocardiography and the Pediatric and Congenital Electrophysiology Society. In addition, The American Institute of Ultrasound in Medicine "supports the value and findings of the statement," and "The Society of Maternal Fetal Medicine supports the statement's review of the subject matter and believe it is consistent with its existing clinical guidelines."

[9] Of note, Class III means that there is either no benefit and/or harm from performing the test and Level of Evidence A means that there have been "multiple populations evaluated" from which to draw the conclusions.

III. Opinions of Plaintiffs' Expert, Nicholas Jewell, PhD

I have reviewed the report of plaintiffs' expert, statistician Nicholas Jewell, PhD. Dr. Jewell argues that a true association has been demonstrated between Zoloft and cardiac defects. In support of his argument, Dr. Jewell states that the studies to date have had limited power to detect an association, that there has been consistent and replicated evidence of increased risk of cardiac malformations, that the effect of confounding is insufficient to explain the apparent association, and that bias from misclassification of medication exposure would mask a true association.

However, his analysis applies inconsistent and improper methodology. Below I detail this unsound methodology.

A. Dr. Jewell Uses Non-Independent Data Sources to Improperly Claim Replication of Study Findings, and Data From One Cardiac Defect to Improperly Claim Consistency With Findings for Other Defects.

Dr. Jewell states that there have been multiple, replicated positive results across multiple studies showing an increased risk of cardiac defects from Zoloft. However, as he notes, some studies use overlapping data: "I do not intend to imply that the results from all of these studies are entirely independent." This is correct; in fact, independent replication of findings is a critical step in evaluating associations, particularly in the setting of the multiple comparisons that have been done in many studies of SSRIs and birth defects.[10]

Dr. Jewell's evaluation of replication and consistency thus has several flaws. First, with respect to the Danish studies (the only studies that demonstrate a statistically significant association between Zoloft and all cardiac defects), Dr. Jewell state that they are "not reporting on the 'same' population." They are, of course, not reporting the exact same population, but they

[10] I discuss the issue and importance of multiple comparisons in my original report.

are also not reporting independent data. The results across these studies clearly include the same patients; this is not independent replication. It does not matter that there are also different patients in the studies; one cannot claim independent replication when the dataset are clearly not independent.

Second, it is important to note that replication should occur using different study populations and designs. Bradford-Hill notes that evaluating for consistency should examine whether an association has "been repeatedly observed by different person, in different place, circumstances and times." Along with the fact that the individual Danish studies do not included independent people, the studies all share similar limitations in their ability to account for confounding. In fact, Jimenez-Solem, the last Danish study, was not done to try to replicate prior studies in the Danish population, but rather to try to address the lingering concerns about uncontrolled confounding in these studies. Thus, the authors explain the purpose and design of their study:

> "None of these [prior] studies have successfully managed to differentiate between the consequences of the drugs themselves and the underlying disease. Given the uncertainty of safety and the common use, we performed a nationwide study of the relationship between SSRI use and congenital malformations with focus on congenital heart defects and comparison with paused use during pregnancy to account for special characteristics of women using antidepressants."

Third, Dr. Jewell uses data from one specific group of defects to purport support for risk for other defects. As I noted in my original report:

> "Finally, different birth defects have different embryological origins and thus one cannot assume that a single drug can cause multiple birth defects of different embryological origins. As Dr. Mitchell notes, "teratogens do not uniformly increase rates of selected defects." (Mitchell 2012) This is important not only in assessing the reliability of claims that a single compound is responsible for a range of birth defects, but also in the proper design and assessment of epidemiological studies. One cannot infer a causal relationship for individual birth defects based on results for other types of birth defects, including categories that combine defects with different embryological origins."

Thus, an association for one category of defects (e.g., septal defects) should not be used as evidence for an association with a different category of defect (e.g., conotruncal defects). For example, Dr. Jewell (page 11) includes results from Colvin for "Other congenital anomalies of the heart" (i.e., excluding all defined categories such as septal defects: OR 3.08; 95% CI: 1.45-6.55[11]) together with other studies reporting on various septal defects as evidence of consistency of results. The results of Colvin are in no way replication of findings for septal defects.

Fourth, Dr. Jewell used data from overlapping outcomes to suggest independent findings. When a set of analyses include overlapping outcomes, one cannot use these overlapping analyses as evidence for replication. For example, an analysis of the outcome, septal defects, would include ASDs, so a separate analysis of ASDs-only cannot be considered independent of the first analysis.

When one considers (as Dr. Jewell acknowledges) that many studies do not present independent data and given the fact that the most recent studies do not replicate earlier findings, there is, indeed, a lack of replication. In fact, when one examines independent studies, there are numerous studies that do not replicate prior associations, including the most recent study by Huybrechts et al. that is itself a study many times larger than all prior cohort studies and the one that best addresses confounding and bias.

[11] As noted in my original report, the results of Colvin et al. are subject to multiple comparisons and lack of adjustment for confounding: they performed at least 81 comparisons of SSRIs and birth defects. They did not adjust for any risk factors (except gestational age) nor for confounding by indication.

B. Dr. Jewell Selectively Relies on Only Certain Portions of Data to Examine Confounding, and Improperly Dismisses or Misrepresents the Objective Evidence Demonstrating That Confounding is a Key Source of False Positive Findings in Earlier Studies.

Dr. Jewell examines several pieces of data in support of his statement that the evidence "strongly refutes that confounding by indication is significantly biasing the increased risks directly associated with Zoloft exposure." However, he dismisses or misrepresents objective data for confounding and selectively reports study findings.

1. Selective Use of Data Distorts the Findings Demonstrating Confounding.

Dr. Jewell states that attempts to adjust for confounding have demonstrated that confounding cannot explain the apparent association between Zoloft and cardiac defects, but considers only some study results and not others. There are, in fact, multiple objective findings that support the importance of confounding in creating false association.

For example, Dr. Jewell fails to note that Huybrechts demonstrated a 14% reduction in the OR with adjustment for confounding, producing an OR of one. Instead, he focuses on only part of the study's adjustment for confounding and not the complete analysis which, as noted above, the authors themselves note as critical ("substantial differences in the characteristics of the patients remained after the cohort was restricted to women with depression.")[12]

He also fails to report the results of Malm where adjustment for confounding reduced the OR for Zoloft and cardiac defects, septal defects, VSDs, transposition of great arteries, and conotruncal heart defects by 19%, 18%, 20%, 17%, and 24%, respectively, all consistent with meaningful and important confounding.

[12] Dr. Jewell also claims that the decrease in the OR in the Huybrechts' depression restricted analysis is "likely the result of a substantial decrease in power." This is objectively not the case as I detail in my review of Huybrechts, above.

He notes that in Ban et al. the OR decreased by 9% and describes this as "minimal." However, this is objective evidence for confounding and, as noted above, Ban's definition of depression would mean that many women who were labeled *depressed, but unmedicated* likely did not actually have depression during pregnancy. Furthermore, Ban finds no statistically significant association between Zoloft and cardiac defects.

Dr. Jewell also makes a direct comparison of Zoloft exposed to paused users in Jimenez-Solem, resulting in a relative risk of 1.59 and 95% CI of 0.80-3.16. (I discuss details of this analysis below). He states that this "is an almost 60% increase in risk, and plausibly supports a doubling or even trebling of the risk[13] entirely contradicting the 'confounding by indication' hypothesis." However, this is, in fact, entirely consistent with confounding by indication; the change in the OR from 2.73 (without direct comparison to paused users) to 1.59 is a 42% reduction in the OR; this is indeed evidence of confounding.

Dr. Jewell notes that Pedersen identified a positive association with Zoloft and septal defects when adjusting for confounding. However, Pedersen adjusted for very few variables (age, calendar year, income, marriage status, and smoking); smoking information was missing in 17% of subjects; and they did not adjust for other potential confounders such as alcohol use, vitamin use, body mass index, opioid use, nor the indication for the drug. This is insufficient to conclude that confounding cannot explain the apparent association and is contrary to what authors, including Pedersen, have stated. For example, Pedersen et al. state "confounding by the indication cannot be ruled out." In fact, the follow-up Danish study (Jimenez-Solem) was conducted because of continued concerns of confounding. Dr. Pedersen was a peer reviewer for

[13] This interpretation also illustrates the problem of selectively examining only evidence that supports one's hypothesis. The results are not statistically significant and the 95% CI also plausibly supports a 20% reduction in risk.

the Jimenez-Solem paper and notes in his review: "I agree with the authors that the association between SSRIs and heart malformation may be caused by systematic error," and the "paper is well written and the results are of both clinical and theoretical importance." Other examples of insufficient adjustment for confounding are noted in my original report.

2. Dr. Jewell Improperly Dismisses or Mischaracterizes Findings Demonstrating Confounding

Dr. Jewell states that studies examining other antidepressants (non-SSRI) have demonstrated "No risk," and that this finding "has been replicated in numerous other studies involving different populations, different methods and different outcomes." This statement is not correct and does not include all of the evidence. Further, Dr. Jewell uses different criteria for evaluating non-SSRIs than he uses for evaluating SSRIs.

a. Numerous Findings Contradict Dr. Jewell's Assertion That Other Antidepressants are not Associated with Increased Risk of Congenital Heart Defects

Reis reports statistically significant ORs for tricyclic antidepressants and cardiovascular defects or VSD/ASD of 1.63 (95% CI: 1.12-2.36) and 1.84 (95% CI: 1.13–2.97), respectively. These findings contradict Dr. Jewell's assertion that such medications are associated with "no risk."

Similarly, in Huybrechts, in the analyses that do not account for depression (which is the appropriate one to use when assessing for false positive associations due to confounding), SNRI antidepressants, buproprion, and other antidepressants are all associated with statistically significant ORs with cardiac defects: the OR (95% CIs) for SNRIs and all cardiac defects, VSDs, and other cardiac defects are 1.51 (1.20–1.90), 1.56 (1.14–2.14), and 1.51 (1.10–2.08), respectively. The OR (95% CI) for buproprion and other cardiac defects is 1.52 (1.14–2.02). The OR (95% CIs) for other antidepressants and all cardiac defects and other cardiac defects are

1.46 (1.16–1.83) and 1.79 (1.34–2.40), respectively. Again, these findings contradict Dr. Jewell's statements.

The only study that Dr. Jewell cites as having demonstrated no risk of cardiac defects from non-SSRI antidepressants,[14] Louik et al., has four odds ratios for cardiac malformations with upper limits of the 95% CI greater than 2 for non-SSRI antidepressants. In other words, the non-SSRI antidepressants have ORs that Dr. Jewell would describe as consistent with a "doubling" of the risk when he examines Zoloft results.

b. Non-SSRI Medications are a Poor Control to Assess Confounding

Even if associations between non-SSRIs and birth defects differ from those for SSRIs, this is not at all unexpected. The indications for non-SSRI and SSRI drugs are not identical and the drugs are prescribed for different indications in practice. For example, by 2005, unlike SSRIs, "TCAs were more commonly used for non-psychiatric indications than for psychiatric indications, especially for sleep- and pain-related reasons." (Patten, "Reasons for antidepressant prescriptions in Canada," *Pharmacoepidemiology and Drug Safety*. 2007).[15]

Accordingly, although analyses of non-SSRIs drugs can be used as an attempt to assess for confounding by indication (and, as demonstrated above, they certainly do provide evidence to support confounding), they are not as useful as analyses that directly adjust for differences in SSRI users themselves (as has been done in recent studies that have demonstrated no association between Zoloft and cardiac defects).

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[14] Dr. Jewell discusses Kornum in his report, but Kornum does not, in fact, present any results for non-SSRI antidepressants and cardiac outcomes (nor does Pedersen).

[15] See also Noordam, "Prescription and indication trends of antidepressant drugs in the Netherlands between 1996 and 2012: a dynamic population-based study," *European Journal of Clinical Pharmacology* (2015)

c. Dr. Jewell Improperly Dismisses Jimenez-Solem's "Paused User" Findings

With respect to the "paused" user group in Jimenez-Solem, Dr. Jewell postulates that this group had an association with congenital malformations because women who restarted SSRIs after pregnancy did so because they had babies with birth defects. It must be recognized that Jimenez-Solem chose to examine women who took SSRIs both before and after pregnancy to try to identify women who had an underlying indication for the drug during their pregnancy. (As discussed above, a woman who simply stops Zoloft months prior to pregnancy is likely to not have depression during pregnancy.) If one does not accept this as a reasonable way to account for confounding, then one must also accept that Jimenez-Solem did not therefore account for confounding by indication in their results. And, because the effects of confounding have both been a concern of all investigators in the field (in fact, the purpose of the Jimenez-Solem study was to assess this in the Danish population) and have been demonstrated to be important in explaining the apparent association between Zoloft and cardiac defects in other studies, the results from Jimenez-Solem that Dr. Jewell relies on are subject to confounding and cannot be taken at their face value.

d. Dr. Jewell's Post-Hoc Reanalysis of Secondary Findings From Huybrechts and His Meta-Analysis are Flawed and Invalid

Dr. Jewell also performs his own re-analyses from Huybrechts and then performs what he calls a "meta-analysis" of his own re-analyses[16] of both Jimenez-Solem and Huybrechts.

With respect to the former, Dr. Jewell's methodology includes several errors.

First, the analyses that Dr. Jewell performs does not include the full adjustment for confounding that the Huybrechts study performs. Such an adjustment cannot be done because

[16] i.e., not the reported results of these studies.

Dr. Jewell's analysis is his own statistical manipulation and does not use the primary data that Huybrechts used in their study.

Second, his underlying premise that this controls for confounding by indication does not consider the important fact discussed above: that depression is not a constant condition and that a woman who stops taking an SSRI is much less likely to have active depression than a woman who is taking an SSRI.

Third, his "paused" group is, in fact, not a paused group. It is a group of women who, by Huybrechts' definition (and in fact by all other studies' definitions), was exposed to Zoloft during the first trimester of pregnancy.

Fourth, the exposed group were those women who required a prescription for Zoloft during the first trimester (i.e., they had to have a prescription filled during the first trimester, and also could have had a prescription that began prior to the first trimester but lasted into the first trimester). That is, these were women who likely had active depression during their pregnancy, and are unlike the group that Dr. Jewell classifies as the "paused" group who, although meeting the authors' definition of exposure to Zoloft during the first trimester, did not receive additional prescriptions during pregnancy.

As a consequence of the errors noted above, the comparison that Dr. Jewell makes is really among women who were all exposed to Zoloft during the 1st trimester and who differed only in whether they were women who required (additional) prescriptions during the first trimester versus women who did not require additional prescriptions. Thus, this analysis includes only women defined as exposed to SSRIs during the first trimester and compares those with a continued indication for the drug with those who may not have had an indication for the drug: That is, this analysis could be further evidence of confounding by indication. In any

event, it does not exclude confounding by indication, does not negate the other objective evidence for this confounding, and does not, as Dr. Jewell asserts, provide "even more convincing evidence" of the risk of Zoloft.

Fifth, the most reliable and thorough analyses of Huybrechts come from their published findings which clearly document the importance of confounding. Dr. Jewell's use of Huybrechts' secondary data, reliance on a false assumption that his analysis adequately accounts for confounding, and performance of an unadjusted analysis, do not in any way add evidence that "strongly demonstrates that the indication for taking Zoloft (including specifically depression because the comparison is made in the depression-restricted cohort), is likely not a substantial confounding factor."

Finally, with respect to Dr. Jewell's purported "meta-analysis" of Huybrechts and Jimenez-Solem, both the "exposed" and "paused" groups that Dr. Jewell develops for the Huybrechts study are different from the "exposed" and "paused" groups in Jimenez-Solem. This makes a meta-analysis of these two studies methodologically inappropriate, regardless of any statistical testing for heterogeneity.

C. Dr. Jewell Misapplies Established Epidemiological Methods to Improperly Dismiss Findings that Contradict his Opinion.

Dr. Jewell states that misclassification of exposure to Zoloft would bias results towards the null and that "potential misclassification bias towards the null generally exceeds the possible confounding and detection biases away from the null." Besides the fact that Dr. Jewell presents no data to provide an estimate of the "size" of this misclassification bias (in contrast with the objective evidence of the size of the bias from confounding), he misapplies several epidemiological principles in his assessment of this bias.

1. Contrary To Dr. Jewell's Assertion, Misclassification of Exposure Does not Necessarily Create a Bias Towards the Null, and Well May Create a Bias Away From the Null.

Dr. Jewell states that "women who received a prescription for a drug may not have actually taken the drug … hence, she would be misclassified as exposed when she was actually unexposed driving the observed association toward no effect (assuming there is an effect from drug exposure)." This statement is inaccurate: this misclassification would not, *a priori*, drive the association towards the null.

As I explained in my prior report: women "who discontinue antidepressants may be more likely to face financial barriers to care, use other psychotropic drugs, abuse alcohol or other drugs, and have multiple comorbidities. (Akincigil A et al, Adherence to Antidepressant Treatment Among Privately Insured Patients Diagnosed With Depression, *Med Care*, 2007 April;45(4): 363–369). These factors could put them at risk of a poor outcome and thus misclassification of these women as 'exposed' (when they are truly unexposed) will create a false association." That is, the bias here is away from the null, creating a false association.[17]

As support for his statement, Dr. Jewell cites Pedersen's results for those women filling two prescriptions versus one prescription as evidence for greater risk with more prescriptions filled (as a marker of better adherence). However, the filling of two prescriptions could simply represent confounding by severity of the indication for SSRIs (as I note in my original report); moreover, the results are based on a total of 5 cardiac defects with exposure to 2 or more prescriptions (making the estimates very unstable). Thus, Pedersen does not provide support for Dr. Jewell's view.

[17] This is a classic example of what may be referred to as an inverse healthy user bias. See, for example, Brookhart MA, Patrick AR, Dormuth C, Avorn J, Shrank W, Cadarette SM, Solomon DH. Adherence to lipid-lowering therapy and the use of preventive health services: an investigation of the healthy user effect. Am J Epidemiol. 2007 Aug 1;166(3):348-54

Notably, Dr. Jewell does not cite Huybrechts, who performed the same analyses of one versus two prescriptions (in a study 54-times larger than Pedersen) and demonstrated no effect of using two or more prescriptions vs one or more prescription during pregnancy on the outcome. The Huybrechts results are thus both contradictory to, and more robust than, the Pedersen results.

2. Misclassification of Exposure Does not Explain Negative Results.

Dr. Jewell also argues that studies that demonstrate a negative association between Zoloft and cardiac defects are attributable to misclassification of exposure to Zoloft and bias towards the null. He states that misclassification of exposure in the Swedish studies that demonstrated ORs less than 1 (i.e., a lower risk with Zoloft) would bias the results towards the null. However, this would mean that the ORs that these studies identified would, if anything, be farther away from 1 then was reported (i.e., an even more "protective" OR).

As noted above, one issue is that misclassification bias would not necessarily be towards the null and data suggest that, if anything, the bias would be towards showing a false positive association. Regardless, even if Dr. Jewell is correct, bias towards the null would mean that the results of the Swedish studies (e.g., the OR of 0.74 in Reis and Kallen) would, if anything, be farther from one (i.e., Zoloft would be "more protective" than the results reported).[18]

[18] Dr. Jewell states that "If a true positive association exists and yet is reduced substantially in size due to significant misclassification of exposure (*e.g.* moving an association with a true Odds Ratio of 2, say, closer to a biased Odds Ratio of 1.3, say) then the effects of chance caused by sampling make it far more likely that a negative association might be observed in contrast to the truth and in studies where exposure misclassification is less of an issue." This is not only completely speculative but inconsistent with the epidemiological concept of bias towards the null. One could use the same speculation and state that the results of studies with ORs greater than one actually reflect true ORs of less than one and are subject to the same effects of bias and chance, thus creating a falsely elevated OR.

3. Dr. Jewell's Analysis of Differential Recall is Erroneous.

Dr. Jewell further states that differential recall in Alwan would be greater than in Louik, biasing the results towards the null to a greater degree in Alwan. There are two errors in this statement.

First, this differential recall would not bias towards the null in either study, but rather would bias the results towards showing a false positive association between Zoloft and cardiac defects (because recall of Zoloft use would be more complete among mothers whose babies have birth defects compared with mothers whose babies do not have birth defects).[19] That is, if one could account for this bias, the OR in Alwan would move further below one (i.e., Zoloft would be "more protective" than the results reported).[20] Dr. Jewell cites Tinker et al.[21] However, Tinker's results mean that the recall bias creating a falsely positive OR would be even more likely in Alwan.

Second, even if one incorrectly assumes that the bias is towards the null, this would have a similar effect and still not explain the results (again, if one could account for the bias, Zoloft would look more protective in Alwan).

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[19] Recall bias in studies of birth defects is a classic example of bias away from the null, discussed in many papers and books, including mine (e.g., Pharmacoepidemiology 5th Edition, p 95, 493-4). See also, for example, Werler MM et al., Reporting accuracy among mothers of malformed and nonmalformed infants, *Am J Epidemiol*, 1989 Feb;129(2):415-21 and Mitchell AA et al., Birth defects related to Bendectin use in pregnancy. 1. Oral clefts and cardiac defects, *JAMA*, 1981 Jun 12;245(22): 2311–14.

[20] Dr. Jewell fails to note this recall bias could also create a false association between Zoloft and septal defects in Louik et al., which also relied on recall. I discuss this important point in my original report.

[21] "increasing [time to interview] may be associated with modest decreases in the quality of data reported during a maternal interview and that this decrease in interview quality might be more pronounced for mothers of control subjects than for mothers of case subjects."

D. Dr. Jewell Uses Inconsistent and Different Standards Depending on Whether the Findings Being Considered Support or Refute His Opinion.

Dr. Jewell applies his own methodology inconsistently in several ways.

1. Improper and Inconsistent Interpretation of Odds Ratios and Confidence Intervals.

The standard scientific methodology is to test the null hypothesis. That is, one assumes that there is no association (the null) and then tests to see if the results obtained reject the null. A 95% confidence interval for an odds ratio (or relative risk) that includes the value of 1 means that the results of a study do not reject the null hypothesis (at the P=0.05 level).

Dr. Jewell improperly focuses only on the upper end of the 95% confidence intervals and then applies different criteria to interpreting studies depending on their results. Specifically, he interprets ORs that are greater than one but not statistically significant (i.e., studies that do not reject the null hypothesis) as "supporting trends," stating that one must not focus solely on statistical significance and that the "Odds Ratio estimate remains the best estimate of the association for the data analyzed." However, when interpreting ORs with values less than one that also are not statistically significant, he focuses not on the ORs, but on the fact that these were "non-significant negative findings" and that their upper bound of the confidence interval was above 1.00, and thus "compatible with an interpretation of increased risk."[22] That is, he only

[22] Dr. Jewell also cites Rothman as support for examining point estimates (ORs). He quotes Rothman's statement that "The results (effect estimates) from the studies could all be identical even if many were significant and many were not, the difference in significance arising solely because of differences in the standard errors or sizes of the studies." This statement does not apply to the Zoloft data: the effect estimates (ORs) are not at all identical across studies and therefore there is clear inconsistency and lack of replication across studies.

focuses on results compatible with elevated risk and dismisses results compatible with reduced risk.[23] In other words, he uses a double standard when assessing the findings.[24]

For example, he includes the OR for septal defects from Kallen et al. (2007) of 1.06 (95% CI: 0.76-1.41) as evidence of a "non-significant supporting trend" but an OR from the same study for "all cardiac defects" of 0.76 (95% CI: 0.47-1.23) as evidence for increased risk because the upper bound of the CI is greater than 1. However, the first OR is most compatible with no risk (OR = 1.06) and the second OR is most compatible with a reduced risk (OR=0.76). In addition, although the upper limit of the CI for the first OR is 1.41, the lower limit of the CI is

[23] Dr. Jewell also states that "Even a study that displays an estimated negative association (reflecting a decrease in risk for births to exposed women) may in fact be compatible with a true positive association if the study has limited power." However, despite the fact that single studies with low power may not be sufficient to determine risk, this does not mean that there is an increased risk that would be identified if only the study were larger. In fact, a study with a negative association is not more likely to be compatible with a true positive association than a true negative association. Interpreting the results in only one way is purely speculative and not consistent with proper methodology.

[24] Dr. Jewell also states in his report that I have relied on non-statistically significant findings to assess for consistency of effects in one of my prior studies and implies that my approach in that study was somehow consistent with his. This is not correct. Dr. Jewell fails to note: (1) the finding of a negative association between SSRIs and MI in that study was in fact an independent confirmation of a statistically significant finding in a prior study (that is, the study was independent replication); (2) the new findings of that study (a relationship between the affinity for the serotonin transporter and the risk of MI) had not been identified previously and therefore, I stated, "Further investigation is needed to confirm these findings"; (3) an independent study was then performed which did not, in fact, confirm these latter findings (as I state in that paper, "No consistent relationship was observed between SSRI affinity and odds of MI"), thus disproving the prior finding. My approach was to consider all the data, perform independent studies, and not examine only the upper limits of confidence intervals. See Sauer WH, Berlin JA, Kimmel SE, Selective serotonin reuptake inhibitors and myocardial infarction, *Circulation*, 2001 Oct 16;104(16):1894-8; Sauer WH, Berlin JA, Kimmel SE, Effect of antidepressants and their relative affinity for the serotonin transporter on the risk of myocardial infarction, *Circulation*, 2003 Jul 8;108(1):32-6; Kimmel SE, Schelleman H, Berlin JA, Oslin DW, Weinstein RB, Kinman JL, Sauer WH, Lewis JD, The effect of selective serotonin re-uptake inhibitors on the risk of myocardial infarction in a cohort of patients with depression, *Br J Clin Pharmacol.*, 2011 Sep;72(3):514-7.

0.76, which is also compatible with a 24% reduced risk. And, further, the lower bound of the second OR is compatible with a 63% reduction in risk. These results should not be interpreted as evidence of reduced risk, just as they should not be interpreted as evidence of increased risk. Taken together, these two results are most compatible with no association.[25]

As another example, Dr. Jewell interprets the results from Malm (OR of 0.93, 95% CI: 0.23-3.76) as being "compatible with a *threefold increase in risk*" but fails to note that the results are also compatible with a 77% reduction in risk.

These are but a few examples. In fact, when one considers study results from independent populations (as one should do when examining data), all studies except for Jimenez-Solem have results that are also consistent with reduced risk. (And, as discussed previously, the ORs from Jimenez-Solem are also subject to confounding that would falsely elevate the OR.)

The flaw in Dr. Jewell's approach to interpreting data, and the inconsistency of his approach, is illustrated in his review of non-SSRI medications. As noted previously, Dr. Jewell states (in his argument for a lack of confounding by indication) that there has been "repeatedly … no such risk" demonstrated from non-SSRI antidepressants. However, there are several statistically significant associations between non-SSRIs and cardiac defects as noted above; most other ORs for non-SSRIs and cardiac defects have values above one; all have upper limits of the 95% CI above one; and many have upper limits of around 2-3.[26] If Dr. Jewell applied his same

[25] Dr. Jewell's approach would mean that a drug that has no true effect on birth defects would have to have data showing it is protective against birth defects (i.e., all data show a statistically significant protective effect) in order to conclude that there is "no association." This is not consistent with proper scientific methodology.

[26] In Jimenez-Solem, the OR for cardiac malformations and tricyclic antidepressants was 1.33 (95% CI: 0.42-4.15), an upper limit of the 95% CI of over 4. In the analyses that do not account for depression in Huybrechts (the appropriate one to use when assessing for false positive associations), six odds ratios for non-SSRI antidepressants and specific categories of cardiac defects all include upper limits of CIs over 2. Although Alwan et al. do not report adjusted odds

criteria for evidence of increased risk from Zoloft to these data, almost all of these results would be consistent with confounding by indication. Yet, Dr. Jewell interprets these data as sufficient to state that there is definitively "no such risk" from these other drugs and thus no evidence for confounding by indication. Thus, Dr. Jewell applies his own criteria inconsistently to these data.

2. Dr. Jewell's Correction for Multiple Comparisons Would Apply Only to the One Study That He Selected.

Dr. Jewell suggests that multiple comparisons could not produce chance findings. In support of his assertion, he performs a statistical adjustment (the Bonferroni correction) to overlapping findings from the Jimenez-Solem study and states that even after making this adjustment for multiple comparisons, the findings remain statistically significant.

Although he does not endorse this method, Dr. Jewell states that it is supportive of his conclusions. However, he does not point out that these data were selected to make his point, yet, had he selected any cardiac defect finding from any other study other than Jimenez-Solem, the finding would be non-significant using the same statistical adjustment.

E. Dr. Jewell Misapplies the Bradford Hill Criteria

As I noted in my original report, "the Bradford Hill criteria are not even applicable to the question of Zoloft and birth defects because there is no demonstrated association between Zoloft and birth defects." Given the data available from the new studies published since this report, it is now even clearer that there is no valid association between Zoloft and cardiac defects. Nonetheless, I would like to correct several errors in Dr. Jewell's discussion and application of the Bradford Hill criteria.

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ratios for non-SSRI antidepressants, they report the exposure data among cases and controls, allowing one to calculate an unadjusted OR of 1.56 (95% CI: 0.96-2.62).

1. Consistency of Findings

As detailed above, Dr. Jewell's overall approach for determining "consistency" of results among studies is methodologically flawed. This includes applying different criteria to studies with positive versus negative results, focusing on only upper limits of 95% CIs, using non-independent data as evidence of replication, and not considering studies' ability to account for confounding.

In this section of his report, Dr. Jewell tries to explain the inconsistent results from several studies as due to misclassification bias. However, this assertion is incorrect. As I note earlier, bias towards the null (e.g., in Kallen and Reis-Kallen) does not explain these findings; in fact, it would make the findings more consistent with a protective effect of Zoloft. Dr. Jewell also states that differential recall in Alwan would lead to bias towards the null. This is the opposite of what differential recall would do in this situation (i.e., this bias would lead to an overestimate of risk, not an underestimate), as noted previously. Thus, these inconsistent results are not explained by misclassification bias. They are, indeed, inconsistent with a relationship between Zoloft and cardiac defects and inconsistent with other studies' findings.

Dr. Jewell notes that "Another method to assess consistency of results from various studies is to perform a meta-analysis. A meta-analysis combines data from two or more independent studies to develop a single conclusion with greater statistical power. This type of analysis can provide a more precise estimate of an overall treatment effect." He then cites the meta-analyses by Myles et al. and McDonagh.

With respect to Myles, Dr. Jewell states: "I do not find Myles (2013) scientifically reliable." He suggests that "the investigators may not have followed their own pre-defined inclusion/exclusion criteria." This is not the case as documented in the Declaration of Dr. Matthew Large, the senior and corresponding author on the study (attached as Ex. B). Dr. Jewell

cites a single sentence in the results section of the Myles paper as evidence that the declaration and the paper "flatly contradict each other." However, he does not cite the actual methods section of the published paper that is entirely consistent with what Dr. Large states in his declaration.[27] In fact, the sentence that Dr. Jewell cites as evidence for outright contradiction references studies that do not actually study SSRIs, have nothing to do with excluding women in the control group, and/or don't even include birth defects.[28] It appears that this sentence is in error. To conclude that the paper is "scientifically unreliable" based on this sentence is unfounded.

Dr. Jewell further criticizes the study because the analysis of Zoloft and cardiac malformation demonstrated heterogeneity. There are two important points to make about this finding: First, heterogeneity suggests inconsistency in findings across studies. Thus, Dr. Jewell illogically includes a meta-analysis that demonstrates inconsistency in results across studies as evidence of "consistency" under the Bradford Hill criterion. Dr. Jewell's repeated statements that there is a consistent observation of increased risk across studies is directly contradictory to what the individual studies and the meta-analysis actually show. Second, the fact that there is unexplained heterogeneity in the study does not warrant exclusion of the study from consideration. The authors appropriately used random-effects models in their summary

[27] The methods section states that the exclusion was for "a control group exposed to any antidepressant medication." This is entirely consistent with Dr. Large's declaration.

[28] Costei et al. is a study of 3rd trimester exposure, not 1st trimester, to paroxetine and, regardless, have no birth defects, so cannot contribute to the meta-analysis. Its control group combines two different groups: one who had all had exposure to paroxetine in the first trimester and one in whom exposure to paroxetine is not reported. Because it does not present results for each control group separately, it is impossible to determine which group had 100% exposure to paroxetine. Queisser-Luft et al. does not include any data on SSRIs. Cole et al. do not provide any data on SSRIs or Zoloft and, in fact, they require exposure to an antidepressant in one of their comparison groups.

estimates, as described in the published paper and also in Dr. Large's declaration. Dr. Jewell suggests that the "summary effect size of 0.93 means that close to half of (future) populations will exhibit increased risk associated with Zoloft exposure, sometimes substantially increased risk (given the large observed heterogeneity)." This would mean that more than half of studies would show a reduced risk associated with Zoloft exposure. That is, it would be more likely that Zoloft reduces risk than increases risk. In addition, there is no evidence that that any studies would show "substantially increased risk" as Dr. Jewell states.

Dr. Jewell also discusses the more recent meta-analysis by McDonagh et al. As noted above, this study demonstrated no association between Zoloft and cardiac defects and accounted for heterogeneity, just at Dr. Jewell states should be done.[29] His criticism is that the section discussing Zoloft and cardiac birth defects consist of "only 3 sentences" and that it is therefore "impossible to fully assess the author's methodology." However, the methods are clearly laid out in their 8 page methods section. Dr. Jewell also discusses double counting, which is resolved in their final analysis as I discuss above. He also criticizes their *a priori* method of excluding certain studies in their analysis of heterogeneity based on ability to adjust for confounding.[30] However, the authors were following their methodology properly and, as noted above, Dr. Jewell's claim that "adjustment for such factors in other studies makes very little difference to the estimated association between Zoloft exposure and the risk of a cardiac birth defects" is both

[29] In fact, the study showed that heterogeneity was, if anything, creating a falsely elevated OR between Zoloft and cardiac defects (the OR before accounting for heterogeneity was 1.08 and after accounting for heterogeneity was 0.76).

[30] The authors state: "Based on input from experts, we identified as key for all outcomes four potential confounding factors to be adjusted for in analyses of observational studies—age, race, parity, and other exposures (e.g., alcohol, smoking, and other potential teratogens). In some cases, additional confounders were considered based on their particular relevancy to specific outcomes. Low or moderate risk-of-bias studies that adjusted for these confounders were considered the best evidence if no RCTs were available."

incorrect[31] and antithetical to the proper method of meta-analysis: one should not decide on the analytic approach based on the results of the studies. Finally Dr. Jewell states that McDonagh's "selected studies are not identified," but one can determine these studies from their report. The studies are: Alwan, Louik, Malm, and Reis. In summary, McDonagh follows proper methodology, examines heterogeneity (again showing inconsistency of results across studies), and demonstrates no relationship between Zoloft and cardiac defects.

2. Specificity

Dr. Jewell states that "specificity requires an examination of: (1) a cause leads to a single effect; and, (2) an effect has one cause." Under this definition for specificity, these criteria are not satisfied (e.g., there are many cases of cardiac defects that occur absent Zoloft use and these defects occurred well before the drug was even on the market). However, Dr. Jewell then states that "I do find support for a specific and consistent association for cardiovascular and septal defects related to Zoloft exposure during early pregnancy." Here he both mixes two different concepts (specific and consistent) and is not, in fact, talking about the criterion of "specificity."

3. Dose-Response

Dr. Jewell suggests that there is "a trend of increased risk with increased dose." He cites Jimenez-Solem, reporting two ORs with widely overlapping CIs and with a statistical test comparing low to high dose of P=0.41. This is not a dose-response and the authors of the paper expressly point out "the lack of relationship between dose and risk." In fact, in Jimenez-Solem, test for an increasing risk with increasing dose (analyzing dose as a continuous variable) "yielded no dose-response association" (Jimenez-Solem), contrary to Dr. Jewell's assertion of "a trend."

[31] See Malm 2011. In addition, as noted above, Pedersen's analysis of smoking was limited by substantial missing data.

Dr. Jewell also claims that Huybrechts supports a dose-response relationship, but the study authors specifically investigated dose-response and concluded that it was not present, reporting: "We did not observe a dose–response relationship either with respect to the first dose or with respect to the highest dose dispensed."

4. Coherence

Coherence, as describe by Bradford-Hill, means that "the cause-and effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease." Here, the disease is cardiac malformations. Dr. Jewell cites data on 3rd trimester exposure to Zoloft and other outcomes as evidence of coherence with 1st trimester teratogenicity. This has nothing to do with coherence. In addition, he cites data on other birth defects as being coherent with Zoloft and cardiac defects, further noting that he has "not been asked to assess whether the above described positive associations are causal." I have been asked to assess this and, as detailed in my prior report, there is no evidence of a causal association between Zoloft and any birth defects.

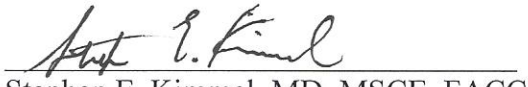
5. Analogy

Dr. Jewell states that the analogy criterion "is clearly satisfied" because "an increased risk for cardiovascular birth defects has been consistently observed for a number of other SSRIs, particularly Paxil (paroxetine) and Prozac (fluoxetine)." However, as I discuss in my original report, there is clear evidence that there is not a class effect among SSRIs. Since that report, the AHA Guidelines (Donofrio (2014)) have reasserted that there is not an analogous effect between paroxetine and other SSRIs.

V. Conclusion

For the foregoing reasons, there is no reliable evidence of a causal association between Zoloft and congenital heart defects. Plaintiffs' expert employed flawed methodologies to reach his opinions, as explained above. I reserve the right to update my opinion if new information becomes available, and I also reserve the right to use graphics and demonstratives to explain and illustrate the subjects discussed in my report. My hourly fee in this matter is $600.

Dated: March 27, 2015



Stephen E. Kimmel, MD, MSCE, FACC, FAHA, FISPE

Schedule of Deposition Testimony in the Past Four Years

- Deposition in Woodson et al. v. Abousy et al., No. CL-2007-5870 (Circuit Court for the County of Fairfax, Virginia)
- Depositions in Fosamax cases on 2/1/2013 and 5/22/2013
- Deposition in Zoloft MDL on 11/26/2013 and Testimony at *Daubert* Hearing on 4/14-15/2014
- Testimony at *Frye* Hearing on 2/17-18/2015
- No trial testimony

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Exhibit A

REPORT FOR ZOLOFT FEDERAL LITIGATION

Expert Report of Stephen E. Kimmel, MD, MSCE, FACC, FAHA, FISPE

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REPORT FOR ZOLOFT FEDERAL LITIGATION

Expert Report of Stephen E. Kimmel, MD, MSCE, FACC, FAHA, FISPE

I am submitting this report in connection with litigation regarding whether Zoloft (sertraline), an antidepressant medication manufactured by Pfizer, can cause birth defects or persistent pulmonary hypertension of the newborn (PPHN). Most of my professional career has been devoted to studying, teaching, and writing on the science of pharmacoepidemiology – the recognized scientific methods by which one can assess whether or not a drug is associated with or can cause a disease. I have authored and edited reference books and textbooks (including perhaps the most comprehensive book in the field), book chapters, and numerous peer-reviewed articles concerning pharmacoepidemiology, and have lectured widely on the subject.

I. Qualifications

I am a Professor of Medicine with tenure in the Department of Medicine at the University of Pennsylvania (Penn), a Professor of Epidemiology in the Department of Biostatistics and Epidemiology at Penn, a Senior Scholar in the Center for Clinical Epidemiology and Biostatistics (CCEB), and a Senior Fellow of the Leonard Davis Institute at Penn. I am also the Acting Director of the Clinical Epidemiology Unit (CEU) of the CCEB and of the Epidemiology Division of the Department of Biostatistics and Epidemiology, and Director of the Center for Therapeutic Effectiveness Research at Penn. I have co-directed Penn's Master of Science in Clinical Epidemiology (MSCE) Program which trains approximately 60 students each year, direct an advanced Master's level course in Cardiopulmonary Epidemiology, and teach epidemiology to both medical students and physicians. In my role in the MSCE program, I have advised pediatric cardiologists who are part of our training programs.

The majority of my time is spent conducting clinical epidemiology research. I have been performing pharmacoepidemiology studies (the study of the use of and the effects of drugs in large numbers of people) for the past 20 years, with a focus on the safety and efficacy of cardiac medications. I have received numerous grants from the National Institutes of Health and other organizations for my research. I am a Fellow of the American Heart Association and its Council on Epidemiology and Prevention, a Fellow of the International Society for Pharmacoepidemiology, and a Fellow of the American College of Epidemiology. I have been elected to the American Society for Clinical Investigation, one of the nation's oldest and most respected medical honor societies. In addition, I have served as an Associate Editor for *Pharmacoepidemiology and Drug Safety*, the official journal of the International Society for Pharmacoepidemiology.

I am an editor of the Textbook of Pharmacoepidemiology (just published in its 2nd edition) which serves as a textbook for graduate trainees in the field, and an editor of the most recent edition of Pharmacoepidemiology, a treatise considered by many to be the definitive reference book in the field. These books include chapters on studying birth defects and details of methodological considerations in interpreting these studies. I have also published on the epidemiology of critical congenital heart disease.

I received my bachelor's degree *Magna Cum Laude* from Princeton University, my MD degree from the New York University School of Medicine (Alpha Omega Alpha honor society), and my MSCE degree from Penn, with a focus in pharmacoepidemiology. I am a board certified cardiologist. I did my internal medicine residency training at Harvard's Brigham and Women's Hospital and my cardiology fellowship at Penn. I continue to perform clinical duties at Penn, including inpatient cardiology consulting and work in the cardiac intensive care unit. I have

treated adults with congenital heart disease as part of my training, and I am also a Fellow of the American College of Cardiology.

For further details please see my attached CV.

II. Materials Reviewed and Relied Upon in Forming Opinions

In forming my opinions in this matter, in addition to the knowledge I bring to this matter based on my work in the field of pharmacoepidemiology, I have reviewed the published medical literature regarding Zoloft and other medications classified as selective serotonin reuptake inhibitors (SSRIs), identified on the attached list of references. I have reviewed the extensive literature that examines the relationship between SSRIs and birth defects or PPHN. In this report, I present all available studies examining Zoloft.

In addition, I present literature examining all SSRI medications as a group, including the SSRI literature cited by plaintiffs' experts, that reveals the methodological limitations arising in such studies of birth defects. While I reviewed and considered in forming my opinions studies that did not include Zoloft but examined other drugs, these studies do not provide data related to Zoloft and therefore are not discussed systematically in this report; nevertheless, as I explain, these studies provide further support for my opinions. Also, although *in vitro* and animal studies can also be performed to examine potential mechanisms of birth defects, such studies by themselves cannot establish causation in humans. The proof of whether or not a substance is a teratogen is determined by human studies.

All of the opinions which I am rendering are generally accepted in the scientific community and are based on scientifically reliable methodology. My prior deposition and trial testimony in the past four years is set forth on the attached schedule. My rate in this matter is $500 per hour.

III. Summary of Opinions

Unfortunately, birth defects are not uncommon. The overall prevalence of major structural or genetic birth defects has been estimated to be 3%. (CDC, Press release: New study finds few risks of birth defects from antidepressant use during pregnancy, 2007 Jun 27). Major cardiovascular congenital defects make up a substantial proportion of these defects, occurring in around 4 to 10 per 1,000 live births in the U.S. (Go AS et al., Heart Disease and Stroke Statistics – 2013 Update, *Circulation*, 2013 Jan 1;127(1):e6-e2455). If one includes all defects (including more minor defects, such as small septal defects), the estimated incidence is much higher, around 75 per 1,000 live births (Hoffman JI, The incidence of congenital heart disease, *J Am Coll Cardiol*, 2002 Jun 19;39(12):1890-900). All of the known birth defects have occurred well before Zoloft (or other SSRIs) were on the market. That is, none are unique to those using SSRIs or Zoloft, and therefore there will be an underlying baseline rate of occurrence of these events among those using SSRIs that would have occurred even in the absence of SSRI exposure. Therefore, a cause and effect relationship cannot be established simply because a woman who was taking Zoloft during pregnancy had a child with a birth defect.

Numerous studies have been conducted to investigate a possible association between SSRIs and birth defects, and substantial data confirm that there is no evidence of a causal link between Zoloft and any birth defects. Moreover, expert regulatory authorities in the United States and other countries have independently assessed the data and have publicly declared that a causal link has not been established, as have independent professional scientific associations. Similarly, the scientific evidence has not established a causal association between maternal Zoloft use and PPHN, which is consistent with the Food and Drug Administration's (FDA) most recent publicly published assessment.

Plaintiff's experts, Anick Bérard, Ph.D. and Michael Jean-Jacques Vekemans, MD, Ph.D., claim that Zoloft causes numerous and diverse birth outcomes. However, their approach suffers from several methodological flaws, and is therefore not scientifically reliable. For example:

- *Overlook Consistent Findings and Conclusions* – The data taken as a whole, including all of the recent and better controlled studies, uniformly do not identify an association between Zoloft and birth defects. Drs. Bérard and Vekemans ignore this key fact.
- *Overlook Contrary Data* – It is not proper scientific methodology to start with a presumed conclusion and then look for studies, or data within studies, that support the presumed conclusion, while ignoring contrary studies and data. In this case, Drs. Bérard and Vekemans selectively focus on isolated studies and data within studies (most of which are not specific to Zoloft) which they claim support their conclusions while overlooking contrary studies and data.
- *Misplaced Reliance on Non-Zoloft Data* – With respect to potential teratogenic effect, it is scientifically suspect to rely on non-Zoloft data, but much of the data relied upon by Drs. Bérard and Vekemans concerns compounds other than Zoloft. It is particularly inappropriate to rely on studies involving other drugs where, as here, those drugs have different chemical structures and pharmacological properties, and there is a large body of epidemiological data specifically examining Zoloft that supports the absence of a class effect.
- *Misplaced and Selective Reliance on Non-Significant, Confounded, or Biased Findings* – Drs. Bérard and Vekemans uncritically and selectively relied on findings likely attributable to chance, confounding, or bias rather than a true association.

IV. Overview of Study Designs

Determining whether a medication has benefit or can cause harm requires very careful research and an appreciation of the strengths and limitations of study designs. Different study designs have different strengths and limitations, and any one study must be judged based on a careful review of these attributes. For purposes of clarity, the following discussion assumes that one wants to examine the effects of a drug on an adverse event.

A. Randomized Trials

Randomized clinical trials designed specifically to address the adverse event of interest can provide the strongest level of support for the effects of a drug. The main reason for this is that randomization should balance the comparison groups on all factors except for the drug exposure, thereby eliminating both measurable and immeasurable forms of confounding. In addition to the benefits of randomization, blinding of patients and investigators to treatment allocation in clinical trials reduces the likelihood of other biases such as ascertainment bias (identifying outcomes differentially based on exposure) and improper recall of drug exposure (as can happen in retrospective study designs). However, randomized trials are not always feasible or ethical, as is typically the case for studies of drug teratogenicity. Therefore, one must often rely on non-randomized study designs, also known as observational studies.

B. Observational Studies

Observational studies do not randomly assign patients to a drug exposure versus no exposure, but rather observe outcomes based on medication use as it occurs in the normal course of events. Because of this design, observational studies cannot ensure balance between exposed and unexposed groups, and therefore uncontrolled confounding (discussed below) can produce inaccurate results.

Observational studies include case-control and cohort studies. Advantages of these study designs include the ability to include a very large number of patients exposed to a drug, allowing one to study rare events with adequate statistical power (thus avoiding Type II error, false negative findings), and providing information from "real world" exposure to the drug (unlike many clinical trials which may recruit selective patient populations). Disadvantages include, but are not limited to, confounding (which can occur if exposed subjects are different from unexposed subjects in ways that can also affect the outcome of interest); selection bias (usually a

concern in case-control studies where the selection of the case group, the control group, or both skews the true prevalence of exposure to the medication in these groups); information bias which includes recall bias (errors in recall of drug exposure by participants) and other inaccurate assessments of drug exposure; ascertainment bias (e.g., differential ascertainment of events among those exposed versus those unexposed to the medication), misclassification of events; interviewer bias (differential prompting of patients to recall medical information); and non-participation bias (nonparticipation of eligible subjects).

One specific type of confounding in observational studies of drug toxicity is called confounding by indication. Confounding by indication occurs when the indication for the drug is also a risk factor for the adverse event of interest.

Another limitation of all statistical analyses is the potential for Type I error (false positive findings), discussed in greater detail below. Type I error can be particularly problematic when one performs numerous statistical tests on the same study data, which increases the potential for Type I error.

C. Case Reports and Case Series

Case reports are individual reports of adverse events among those using a drug, and case series are collections of such reports. With rare exception (that does not apply to this case), they cannot be used to determine causality because they cannot be used to estimate the rate of adverse events nor the risk of these events relative to events spontaneously occurring and unrelated to drug exposure. This is because they do not include a comparison control group of unexposed individuals nor do they provide an estimate of the total number of events occurring or the number of exposed individuals. Thus case reports are usually useful for generating hypotheses only. This is particularly true for birth defects (which are not uncommon among women not exposed to SSRIs).

D. Spontaneous Adverse Event Reporting

Adverse event reports (AERs), reports to FDA's spontaneous reporting system, are in essence case reports as well, albeit often without the same level of detail of information as case reports. A reportable adverse drug effect is "any adverse event associated with the use of the drug in humans, whether or not considered drug related…." (Baum C et al., The spontaneous reporting system in the United States. In: Strom BL, ed., *Pharmacoepidemiology*, 2nd ed., New York, NY, John Wiley & Sons, 1994:125-37). As such, the FDA's reporting system can serve as a signal of a possible problem and is primarily useful to generate hypotheses. In the case of birth defects (which are not uncommon among women who do not use SSRIs), adverse event reports, like case reports, cannot be used to determine causality. There are several well-known limitations to this reporting system that further prevent it from being used to determine causality.

First, in order to be reported, an event must be identified, attributed to the drug (e.g., by the patient or health care provider treating the patient, a lawyer, or anyone else), and then reported to the FDA. As a result, comparative studies of reporting rates between different drugs can be misleading. The degree of reporting can vary between drugs, thus creating differences among drugs that are not related to true differences in risk. For example, a newly named brand of penicillin appeared to be causing a four-fold increase in the risk of hemorrhagic diarrhea after release on the market relative to a previously released brand of penicillin. However, the two forms of the drug were produced in the same factory from the same batch and differed only in form, product name, and drug company. Further analysis revealed that there was no difference in risk between the products. The reason for the apparent increased risk from this "new product" was that the physicians and countries using the new brand were more likely to report adverse events in general than physicians or countries using the older brand of penicillin (Wiholm BE

et al., Spontaneous Reporting Systems Outside the United States. In: Strom BL, ed., *Pharmacoepidemiology*, 2nd ed., New York, NY, John Wiley & Sons, Inc., 1994:139-55). Reports of an adverse event in the medical community or lay press can also increase reporting (publicity bias), again unrelated to an actual risk from the drug.

Second, spontaneous reporting does not allow for a determination of the incidence (risk) of adverse events. Because the true number of events is unknown and because the number of exposed individuals is unknown, the true risk (number of events divided by number of exposed) is unknown. As stated by two authors from the FDA: "One of the greatest limitations of any spontaneous reporting system – and perhaps the one accounting for the greatest misuse of ADR reporting data – is its inability to provide *incidence rates*.…" (Baum C et al., The Spontaneous Reporting System in the United States. In: Strom BL, ed., *Pharmacoepidemiology*, 2nd ed., New York, NY, John Wiley & Sons, 1994:125-37).

Third, because there is often an underlying baseline risk of disease unrelated to exposure to a product, there will be events reported in people exposed to that product that are in no way associated with use of the product. This is particularly problematic if a drug is used in those at highest risk of the adverse event of interest. In addition, other confounding factors are often present (e.g., other medication use, lifestyle factors), making it extremely difficult to determine if an event truly is related to the exposure of interest.

V. Selective Serotonin Reuptake Inhibitors (SSRIs) and Birth Defects

A. Overview

With this background in basic epidemiological principles, I reviewed the published, observational studies on birth defects and SSRIs (there are no published randomized trials for the reasons stated previously). As noted above, I focus on studies examining Zoloft, the actual drug at issue here. Nonetheless, I also present literature examining all SSRI medications (including

studies cited by plaintiffs' experts) which reveal the methodological limitations arising in such studies of birth defects. Also as previously noted, like the studies on Zoloft, the studies on SSRIs overall (without differentiating drug) show no significant association with birth defects. It is particularly inappropriate to rely on studies involving other drugs where, as here, those drugs have different chemical structures and pharmacological properties, and there is a large body of epidemiological data specifically examining Zoloft and also supporting the absence of a class effect. (*See* Mitchell 2012.)

Many of the limitations discussed generally in Point IV above are of particular concern in studies of SSRIs and birth defects. There are a number of biases and confounding factors present in such studies that tend to bias the studies toward reporting an association when no true association is present. I will review several of these here and provide some examples. In my review of individual studies, I will highlight these limitations as appropriate to the studies being reviewed.

1. Type I and Type II Error

Type I error due to multiple comparisons can lead to false positive relationships in studies of birth defects. Type I error is typically set at 0.05, corresponding to a P-value threshold of 0.05. This represents the probability of finding a result as extreme or more within the study purely due to chance. However, multiple testing will increase the likelihood of obtaining a result that appears to be inconsistent with chance when it really is due to chance. This is particularly problematic in the studies of SSRIs and birth defects because investigators will often perform numerous statistical tests on the same dataset, increasing the chances of false positive findings.

For example, in Alwan the investigators report on 265 statistical comparisons in Table 2 (Alwan S et al., Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects, *N Engl J Med*, 2007 Jun 28;356(26):2684-92). With this many comparisons, under the

null hypothesis (that is, there are no true relationships between the drugs and any of the birth defects) there is an almost 100% chance that one will report a "statistically significant" relationship (here defined as a P-value less than 0.05) simply by chance. (The formula for this calculation is: $[1-(1-\alpha)^n]$ where α is the type I error rate (here 0.05) and n is the number of statistical tests.) Of note, investigators typically do more statistical tests than they are able to report in a published study.

A related concept is the 95% confidence interval (95% CI). A 95% confidence interval for an odds ratio (OR) or relative risk (RR) that excludes the value of 1 will usually be associated with a P-value of less than 0.05. The width of the CI can provide some estimate of the precision of the estimate of the OR or relative risk (RR).

Type II error (false negative results) can also occur, particularly with small numbers of outcomes. However, one should not assume that, just because a study is small, it has failed to find a true association. Scientific studies first assume the null, that there is no association, and then test to see if the results obtained are consistent with the null.

2. Confounding

Confounding is another concern in these studies that tends to bias them toward reporting an association when in fact no true association is present. Confounding can occur if exposed subjects are different from unexposed subjects in ways that can influence the occurrence of the outcome. For example, women who use SSRIs or have indications for SSRIs (e.g., depression) are substantially different than women who do not use SSRIs. In studies of SSRI use in pregnancy, women who use SSRIs are more likely to smoke, be of older age, have less education, live alone, have fewer health care encounters, not take their medications correctly (poor adherence), have poor nutrition, have low birth weight babies at lesser gestation ages, use multiple other drugs (such as opiates, non-steroidal anti-inflammatory drugs, thyroid drugs,

anticonvulsants, neuroleptics, sedatives, hypnotics, and other psychotropic drugs), not use multivitamins and folic acid, be unmarried, have multiple chronic diseases including diabetes and hypertension, and have babies with fetal alcohol spectrum disorder and withdrawal symptoms from drugs of addiction and therapeutic medications (Källén 2006) (Källén 2007) (Källén 2010) (Margulis 2013) (Malm 2011) (Colvin 2011) (Alwan 2007).

These factors could all also increase the risk of birth defects or other adverse events that may increase the detection of birth defects. In the absence of being able to account for these factors, associations between SSRIs and birth defects may be spurious. Further, ascertainment of these and other risk factors may be incomplete, resulting in incomplete control for confounding. For example, illicit drug use is often substantially underreported, even if information is collected from medical records or patient interviews (Mitchell 2012).

One type of confounding that is particularly problematic is called "confounding by indication." Confounding by indication occurs when the indication for the drug is also a risk factor for the adverse event of interest. Depressed women (and particularly women who take SSRIs) have certain behaviors and characteristics that have been linked to increased risk of birth defects or other peripartum complications. Thus, comparing women on SSRIs with women who are not being treated with SSRIs for depression, can lead to apparent associations between SSRIs and adverse events that are due to the underlying depression, and not the drug. Because depression can be linked with many different risk factors and/or habits (such as nonadherence with medical advice, diet, etc), even adjusting for typical risk factors (such as smoking and alcohol use) may not fully account for confounding by indication. Moreover, limitations in available data may make it impossible to control for all potential confounders.

3. Bias

Ascertainment Bias: Differential ascertainment of events is one form of bias that can also create false associations. This occurs if those exposed to a drug are more likely to have a diagnosis of a condition made than those who do not use the drug, even in the absence of a true relationship between the drug and the condition. Women using SSRIs and their infants have been shown to undergo more prenatal and postnatal testing than those who do not use SSRIs. Women using SSRIs are more likely to undergo fetal ultrasounds during pregnancy, and the infants of these women are almost twice as likely to undergo echocardiograms as infants of women who do not use antidepressants (Bar-Oz et al., Paroxetine and congenital malformation, *Clin Ther*, 2007 May;29(5):918-26). Such differential testing could uncover more birth defects, particularly those that would not otherwise be clinically evident, among SSRIs users, even if SSRIs did not increase the risk of birth defects (Koren G, The effect of ascertainment bias in evaluating gestational antidepressant exposure, *J Popul Ther Clin Pharmacol*, 2011;18:e174-75). This could be important, for example, for atrial or ventricular septal defects which often do not manifest in a clinically obvious manner immediately after birth and often resolve spontaneously (Koren 2011).

It is also important to note that SSRIs may be used for conditions other than depression, most notably anxiety disorders (Margulis AV et al., Use of selective serotonin reuptake inhibitors in pregnancy and cardiac malformations, *Pharmacoepidemiol Drug Saf*, 2013 Sep;22(9):942-51). (Paroxetine was the first SSRI approved, in 2001, for generalized anxiety disorder.) Such conditions could also lead to increased screening of pregnant women and their newborns. In addition, infants born to mothers using SSRIs may exhibit characteristics such as tachypnea (rapid breathing), irritability, and withdrawal symptoms from drugs of addiction and therapeutic medications, all of which can lead to increased clinician scrutiny, more diagnostic

tests, and thus identification of defects that would not otherwise be detected in women who do not use SSRIs.

Another type of ascertainment bias can occur if those with the outcome and the exposure are not identified. For example, some have hypothesized that if pregnant women using a particular medication are at increased risk of spontaneous abortions due to certain birth defects themselves, then including only live births could potentially diminish a true association between the drug and certain outcomes. However, it is important to note that many birth defects typically do not lead to spontaneous abortions. Thus, it is unlikely that studies of these birth defects are biased by spontaneous abortions. (See discussion on Pages 50-51 below.)

Information Bias – Recall: Recall bias is another concern, particularly in retrospective studies in which women provide self-reported information about their prenatal exposures after their babies are born, which would tend to bias studies toward reporting an association when no true association was present. The mother of an infant with a birth defect "may be more likely to recall every possible act, event, and drug exposure in pregnancy." (Werler MM et al., Reporting accuracy among mothers of malformed and nonmalformed infants, *Am J Epidemiol*, 1989 Feb;129(2):415-21). This tendency is reinforced by repeated inquiries from physicians, nurses, genetic counselors, and relatives, as well as by media and legal attention on the subject of potential drug-induced birth defects. Thus, in a setting where drug exposure is in fact identical among mothers of normal and malformed infants, one might predict that recall of exposure will be more complete among the latter than among the former, creating a false association between the drug and the birth defect (Mitchell 2012). Such bias has been identified as a cause of prior false associations of medications with birth defects (for example, bendectin) (Mitchell AA et al.,

Birth defects related to Bendectin use in pregnancy. 1. Oral clefts and cardiac defects, *JAMA*, 1981 Jun 12;245(22): 2311–14).

Information Bias – Medication Adherence: Another type of information bias is produced by poor medication adherence. Despite reported use of medications (by self-report, medical records, or pharmacy data), patients may not take their medications. If those who do not take their medications are also at higher risk of the outcome, then this misclassification will produce a bias towards showing a false association between the drug and the outcome. For example, those who discontinue antidepressants may be more likely to face financial barriers to care, use other psychotropic drugs, abuse alcohol or other drugs, and have multiple comorbidities. (Akincigil A et al, Adherence to Antidepressant Treatment Among Privately Insured Patients Diagnosed With Depression, *Med Care*, 2007 April;45(4): 363–369). These factors could put them at risk of a poor outcome and thus misclassification of these women as "exposed" (when they are truly unexposed) will create a false association.

4. Class Effect

In studies of teratogenicity, a class effect cannot be assumed for drugs, even where they have similar therapeutic use and chemical structure (Mitchell 2012). It usually cannot be known whether the chemical structure common to a drug class is responsible, or whether the components that differentiate one class member from another are responsible, for teratogenesis. For example, thalidomide and glutethimide are both glutarimides, and both are sedative/hypnotics. Nevertheless, despite their structural and clinical similarities, thalidomide is clearly a high-risk teratogen but glutethimide is not (Heinonen OP et al., Sedatives, tranquilizers, and antidepressant drugs. In: Kaufman DW, ed., *Birth Defects and Drugs in Pregnancy*, Littleton, MA, Publishing Sciences Group, 1977:335-44).

As Dr. Mitchell notes in a section of his book chapter entitled "The fallacy of 'class action' teratogenesis," class action "cannot be assumed to hold when the adversity at issue is teratogenesis" (Mitchell 2012). This particularly applies to SSRIs, which have parent compounds and metabolites with differing pharmacological characteristics (Louik C et al., First-trimester use of selective serotonin-reuptake inhibitors and the risk of birth defects, *N Engl J Med*, 2007 Jun 28;356(26):2675-83).

5. Specificity

Finally, different birth defects have different embryological origins and thus one cannot assume that a single drug can cause multiple birth defects of different embryological origins. As Dr. Mitchell notes, "teratogens do not uniformly increase rates of selected defects." (Mitchell 2012) This is important not only in assessing the reliability of claims that a single compound is responsible for a range of birth defects, but also in the proper design and assessment of epidemiological studies. One cannot infer a causal relationship for individual birth defects based on results for other types of birth defects, including categories that combine defects with different embryological origins.

B. Summary of Methodological Issues Related to Studying SSRIs and Birth Outcomes

Thus, there are numerous potential limitations and biases that have been recognized in studies of birth defects, including specifically studies involving Zoloft and birth defects, which would tend to bias the results toward finding an association when none in fact is there. That is, even though a study may report a relative risk greater than 1.0 for Zoloft use and a birth defect, this association may be false. It is therefore critically important to evaluate each study to determine the limitations and biases that may skew the results (as is often recognized by the study authors), rather than simply take the results at face value.

VI. Studies Examining Relationship Between SSRIs and Birth Defects, Focusing on Zoloft

Below I discuss the published papers that examined Zoloft specifically and/or provide important data concerning methodological issues within the literature. As noted previously, I considered all of the published literature in forming my opinions and the inclusion of any additional papers from the literature not specifically referenced in the section below would only strengthen my opinions.

I have organized this section by groups of studies that examined SSRIs in different geographic areas. Using this organization is important because it addresses two important issues: First, many of the studies from the same geographic region include overlapping data; one cannot consider data from these studies as independent verification of their own results. In other words, overlapping data is a form of "double-counting." Equally important, it also can propagate earlier errors into subsequent studies. For example, if an association is identified by chance or due to error in a population in one study, it would be expected to be seen again if that population was studied again; this does not mean that the association has been validated. In addition, one must consider the specifics of each study's design in order to appreciate what the results may or may not mean. Second, the progression of studies from the same geographic region can identify the methodological and statistical limitations of earlier study results, such as confounding, bias, and Type I error. After each of the groups of studies, I summarize the overall conclusions.

A. Swedish Population Studies

1. Källén

Källén published a study in 2007 using a Swedish population from July 1, 1995 through 2004 (Källén et al, Maternal use of selective serotonin re-uptake inhibitors in early pregnancy and infant congenital malformation, *Birth Defects Res A Clin Mol Teratol*, 2007

Apr;79(4):301-08).[1] The study performed at least 39 different comparisons. There were no significant associations between Zoloft and any of the congenital malformations examined, including overall malformations (OR 0.78, 95% CI: 0.61-1.00), any cardiac malformations (OR 0.76, 95% CI: 0.47-1.23), and VSD and/or ASD (OR 1.06, 95% CI: 0.63-1.77). Only paroxetine demonstrated a statistically significant relationship with overall cardiac malformations, and, as discussed below, the difference between paroxetine and other SSRIs was statistically significant in analyses of this population, consistent with the absence of a class effect (Reis M Källén & Källén B, Delivery outcome after maternal use of antidepressant drugs in pregnancy, *Psychol Med*, 2010 Oct;40(10):1723-33) (Källén B, Letter to the Editor: Antidepressant drugs during pregnancy and infant congenital heart defect, *Reprod Toxicol*, 2006 Apr;21(3): 221–22). Of note, there was no significant association between Zoloft and craniostenosis (i.e., craniosynostosis) or abdominal wall defects (gastroschisis and omphalocele), suggesting false positive results in Alwan, as discussed below (Alwan S et al., Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects, *N Engl J Med*, 2007 Jun 28;356(26):2684-92). As the authors noted, they could not account completely for many confounders including confounding by indication, differential screening, or multiple

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[1] In total there were 6,481 women reporting SSRI use from July 1995 through 2004. Information on medication use was collected during the first trimester in most women, limiting the possibility of recall bias. Adjustment was made for some potential confounders (year of birth, maternal age, parity, smoking, and 3 or more previous miscarriages), but not for other differences between SSRIs users and non-users identified in the study including other medication use and body mass index. SSRI users were more likely to use some medications such as NSAIDs, opioids, sedatives, hypnotics, anticonvulsants, drugs for migraine and stomach ulcers, oral contraceptives, thyroid hormones, neuroleptics, and antiasthmatics; and less likely to use others such as multivitamins and folic acid. They also did not adjust for other lifestyle, socioeconomic factors, or indication for SSRIs.

comparisons in their study. Källén noted: "Nothing is known about the medical condition that was the reason for drug intake. This leaves the underlying disease as a confounder, which is very difficult to eliminate." (Källén et al, Maternal use of selective serotonin re-uptake inhibitors in early pregnancy and infant congenital malformation, *Birth Defects Res A Clin Mol Teratol*, 2007 Apr;79(4):301-08)

2. Reis

A follow-up study, again using the Swedish population, examined outcomes from July 1995 to 2007, included 57% more women with exposure to SSRIs than in their prior study (Reis M & Källén B 2010). They noted that women who used SSRIs had many more comorbidities, other exposures, and delivery complications than non-users. All of these factors could lead to falsely elevated odds ratios between SSRIs and outcomes through either confounding or enhanced detection of birth defects.[2] The authors performed at least 39 comparisons. Zoloft was not associated with relatively severe malformations (OR 0.99, 95% CI: 0.81-1.21), any cardiovascular defects (OR 0.74, 95% CI: 0.50-1.09), hypospadias (OR 0.89, 95% CI:0.38-1.75), or VSD/ASD (OR and CI not presented in the paper). It is important to note that in this study, with 52% more cardiovascular defect cases among Zoloft users than the prior Swedish study by Källén, there still was no demonstrated significant relationship between Zoloft

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[2] Women who used SSRIs were more likely to be older, of lower parity, smokers, non-cohabitating, working less full-time, and with higher BMI. They also were more likely to use numerous other drugs with a very high use of sedatives and hypnotics and also neuroleptics, anti-migraine drugs, and anticonvulsants. Antidepressant users were also more likely to have numerous maternal delivery diagnoses, including gestational diabetes, pre-eclampsia, placenta previa, placenta abruption, bleeding, and caesarian sections. Infants born to mothers on antidepressants were also more likely to have hypoglycemia.

and cardiovascular birth defects (Källén 2007). Only paroxetine was associated with a statistically significant OR for cardiovascular defects, and the authors note that the difference in cardiovascular defect ORs were statistically significant between the four SSRIs studied; that is, there was statistical evidence that SSRIs differed in their effects on cardiovascular birth defects and did not act as a class.

3. Summary of Swedish Studies

Thus, taken together, the Swedish studies did not identify an association between Zoloft and congenital malformations. In addition, there is evidence for a difference among SSRIs in terms of potential teratogenic effect, consistent with the fact that one cannot assume a class effect for teratogenicity. These studies also demonstrate the marked differences between women who use antidepressants compared with women who do not use these drugs. The authors note that these differences could confound their analyses, that even adjustments for those measured confounders may have been incomplete, and that other confounders were not studied (such as alcohol use and the underlying condition for which the drugs were prescribed). As the Reis and Källén 2010 study noted: "The most difficult confounder, much discussed in the literature, is the underlying pathology, notably maternal depression. We cannot distinguish between the effects of depression and drug treatment of depression." They also note the Type I error that could be introduced by multiple comparisons.

B. Finish Population Studies

1. Malm 2005

Malm, in 2005, studied a Finish population using several available databases and compared women exposed to SSRIs at various times during pregnancy with women who did not use any SSRIs during or one month before pregnancy (Malm et al., Risks associated with selective serotonin reuptake inhibitors in pregnancy, *Obstet Gynecol*, 2005 Dec;106(6):1289-96).

They did not identify an association between major malformations and Zoloft used in the 1st trimester.[3]

2. Malm 2011

Malm published an updated analysis of the Finnish cohort using 1996-2006 data, overlapping with data presented in their 2005 study (Malm 2011).[4] In this study they examined specific malformations and found no significant associations between Zoloft and any malformations, including overall malformations, major cardiovascular anomalies, atrial septal defects, ventricular septal defects, right ventricular outflow tract obstructions, isolated cases of transposition of great arteries, conotruncal heart defects, left ventricular outflow tract defects, central nervous system defects, neural tube defects, respiratory defects, cleft lip with or without cleft palate, digestive system defects, urogenital defects, musculoskeletal defects, omphalocele, or craniosynostosis. This study also illustrates how confounding can bias the results toward reporting an association where none is in fact present. When Malm adjusted for the confounders measured, the odds ratio for SSRIs overall and major cardiovascular defects dropped from 1.29 to 1.09, a substantial 15% reduction in the odds ratio. These results are not surprising given that

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[3] The study did not adjust for underlying depression or other psychiatric disease, nor did it adjust for several other potential confounders such as body mass index, alcohol use, illicit drug use, and nonprescription medication use. The study also did not examine specific malformations. The study did, however, include pregnancy terminations due to fetal malformations and all neonates were examined by a pediatrician prior to hospital discharge, potentially mitigating detection bias. In addition, the authors noted that the study was powered to detect a two-fold risk.

[4] They adjusted for only maternal age, parity, year of pregnancy, smoking, purchase of other reimbursed psychiatric drugs, and maternal diabetes. They did not adjust for BMI, indication for SSRIs, other health-related factors (e.g., vitamin use, diet, etc), alcohol use, illicit drug use, or nonprescription medication use.

women with SSRI purchases had substantially more comorbidities and other exposures than women without SSRI purchases.[5] Moreover, Malm's adjustment did not account for all potential confounders in the study. For example, Malm reported that fetal alcohol spectrum disorders were almost 10 times more common among SSRI users, demonstrating "substantial maternal alcohol consumption" among these women, a potential confounder that was not accounted for. As Malm later noted, "[O]ne of the major problems in pharmacoepidemiologic studies is confounding by indication. Untreated maternal depression has a negative impact on several pregnancy outcome measures and is difficult to control for in epidemiological study settings." (Malm H et al., Prenatal exposure to selective serotonin reuptake inhibitors and infant outcome, *Ther Drug Monit*, 2012 Dec;34(6):607-14).

3. Summary of Finish Studies

The studies by the Malm group provide evidence that there is no association between Zoloft and any malformation, including all major cardiac malformations and septal defects.

C. Western Australian Population Studies

Colvin et al. published a study using data from Western Australia from 2002 to 2005 (Colvin L et al., Dispensing patterns and pregnancy outcomes for women dispensed selective serotonin reuptake inhibitors in pregnancy, *Birth Defects Res A Clin Mol Teratol*, 2011 Mar;91(3):142-52). They performed at least 81 comparisons of SSRIs and birth defects. They only adjusted for gestational age and not for other risk factors nor indication for SSRIs. There

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[5] Women who purchased SSRIs were less likely to be married, twice as likely to smoke or to be entitled to special reimbursement because of chronic disease, and 20-times more likely to have purchased other psychiatric medications than women who did not purchase SSRIs.

were no statistically significant relationship between Zoloft and multiple types of birth defects, including cardiovascular defects as a group; bulbus cordis anomalies of cardiac septal closure; VSDs; ASDs; hypoplastic left heart; anomalies of the pulmonary valve; patent ductus arteriosus; nervous system defects; eye anomalies; ear, face, and neck anomalies; gastrointestinal defects; urogenital defects; musculoskeletal defects; and hypospadias. The study reported an odds ratio of 3.77 (95% CI: 1.19-11.95) for Zoloft and "respiratory system defects," but this was based on fewer than 5 exposed cases and was not replicated in other studies, including the Malm 2011 study. Finally, as the authors noted, "we did not find any statistically significant increases [from Zoloft] of any specific birth defects at the fourth digit level of the British Pediatric Association." (Colvin 2001)

D. United States Population Studies

1. Alwan

Alwan published a case-control study in 2007 using data from the U.S. National Birth Defects Prevention Study (Alwan S et al., Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects, *N Engl J Med*, 2007 Jun 28;356(26):2684-92).[6] This study – which was performed by researchers including scientists at the CDC – found that with respect to Zoloft, there was no association with 18 pooled birth defects, cardiac defects, or non-

[6] The study included elective terminations in 5 states and infants dying at greater than or equal to 20 weeks gestation in 6 states. Although the study adjusted for several potential confounders, including smoking and obesity, it did not account for indication for the drugs (i.e., confounding by indication). Consistent with this possibility, there were more reports of non-SSRI antidepressants among cases than controls. Information on exposure was collected from the mothers after delivery, and thus is subject to possible recall bias. Also, only 71% of case mothers and 69% of control mothers participated in the study, potentially introducing selection bias.

cardiac defects. This includes numerous individual cardiac defects (conotruncal defects, septal heart defects, and right ventricular or left ventricular outflow track obstructions), spina bifida, cleft lip, cleft palate, anorectal atresia, hypospadias, transverse limb deficiencies, craniosynostosis, omphalocele, diaphragmatic hernia, and gastroschisis. The study reported an OR for anencephaly and Zoloft use of 3.2 (95% CI: 1.1-9.3). This was identified among 265 comparisons overall and at least 15 comparisons with Zoloft, and the study did not account for multiple comparisons. The authors note that the few findings reported in the study had not been seen in several prior studies and that, "[b]ecause of the large number of comparisons evaluated in our analysis, it is likely that some of the observed associations reflect chance variation." The authors also cautioned: "analysis of other data sets are warranted to replicate our findings." Of note, a paper published in the same journal (discussed below) failed to identify an association between Zoloft and neural tube defects (which include anencephaly). Alwan also grouped together the 3 defects that they identified in their initial analyses (anencephaly, craniosynostosis, and omphalocele) and there was no statistically significant association reported for Zoloft.[7] Accordingly, in announcing the results of the study, the CDC reported: "Overall, our results are generally reassuring with respect to the use of antidepressants during pregnancy." (CDC, Press release: New study finds few risks of birth defects from antidepressant use during pregnancy, 2007 Jun 27)

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[7] These latter analyses are not only subject to all of the limitations of the study noted above, but are also data-derived (i.e., determined by the results obtained in the study analyses) and thus should not be considered hypothesis-testing results.

2. Louik

In the same issue of the same journal, Louik published the results of case-control study using the Slone Epidemiology Center Birth Defects Study (Louik C et al., First-trimester use of selective serotonin-reuptake inhibitors and the risk of birth defects, *N Engl J Med*, 2007 Jun 28;356(26):2675-83).[8] The authors performed 42 initial and 66 additional exploratory comparisons, subjecting the study to identify associations purely by chance. There was no statistically significant association between Zoloft and numerous birth defects, including all cardiac malformations, conotruncal defects, left ventricular outflow tract defects, right ventricular outflow tract defects, cleft lip with or without cleft palate, pyloric stenosis, renal-collecting-system defects, hypospadias, clubfoot, cleft palate alone, undescended testis, neural-tube defects, diaphragmatic hernia, and craniosynostosis. The study reported an odds ratio of 5.7 (95% CI: 1.6-20.7) between Zoloft and omphalocele. However, along with concerns of multiple testing creating a false positive result, the authors note that this was based on a total of 3 exposed cases, making this estimate very unreliable. As the authors note: "We included results based on small numbers of exposed subjects in order to allow other researchers to compare their observations with ours, but we caution that these estimates should not be interpreted as strong

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[8] This study compared 9,849 cases (those with birth defects) with 5,860 controls (those without birth defects) and collected self-reported medication use based on interviews with women after they had their babies (up to 6 months after delivery). This approach subjects the study to the potential for recall bias. In addition, only 61.7% of mothers with affected infants and 59.9% of control mothers participated, subjecting the study to non-participation bias. The analyses adjusted for age, maternal race or ethnic group, education, year of last menstrual period, study center, 1st trimester smoking and self-reported alcohol use, history of birth defect, body mass index, parity, seizures, diabetes, hypertension, infertility, and folic acid use. Other possible confounders such as other medication use, lifestyle factors, and indication for SSRIs were not included.

evidence of increased risks." The study reported an odds ratio of 4.4 (95% CI: 1.2-16.4) for Zoloft and septal defects, but this was among 30 comparisons for antidepressants and just cardiac defects and among 108 comparisons overall. Equally important, there was no association between Zoloft and septal defects in Alwan (OR 0.7, 95% CI of 0.3 1.5). In exploratory analyses, among 66 comparisons, the study reported an odds ratio of 4.4 (95% CI of 1.2-16.4) for Zoloft and anal atresia and an odds ratio of 3.9 (95% CI of 1.1-13.5) for Zoloft and limb-reduction defects. The authors cautioned that these "previously unreported associations . . . warrant particularly cautious interpretation," and "warranted further exploration" and "further studies." They also noted, "The possibility that chance accounts for some or all of these results cannot be ruled out, especially in view of the many comparisons that were made in these analyses" Of note, there were no such associations in Alwan et al. (specifically anal atresia or limb deficiencies), and these findings were not confirmed in other studies as well. None of these exploratory findings were hypothesized in advance, and thus they are likely to be chance findings due to multiple comparisons. Overall, this study does not support an association between SSRIs in general and Zoloft specifically with birth defects. Also, as noted by the authors, "Our analysis did not confirm previously reported associations between overall use of SSRIs and craniosynostosis, omphalocele, or heart defects as a group."

3. Summary of U.S. Studies

Thus, overall, Alwan and Louik do not provide evidence for an association between Zoloft and birth defects. In fact, these studies highlight the problems of multiple comparisons and unstable estimates with small numbers of events. Findings in one study are often not replicated in another study. For example, the findings for Zoloft and septal defects, anal atresia, and limb-reduction defects in Louik were not verified in Alwan, and the finding for Zoloft and anencephaly in Alwan was not verified in Louik.

In addition, like the Finnish studies, the U.S. studies failed to control for confounding by indication, which would tend to bias the study toward finding an association when none in fact exists. As Louik noted: "A major potential confounder is the effect of depression itself, unrelated to drug treatment." Alwan similarly noted: "An important limitation of this study is our inability to separate the effect of maternal SSRI use from that of the underlying depression."

E. Danish Population Studies

There is a series of four studies that all used a cohort of Danish women (Wogelius 2006) (Kornum 2010) (Pedersen 2009) (Jimenez-Solem 2012). The most recent study, conducted by Jimenez-Solem (2012), provides evidence that confounding by indication is present in studies that examine SSRIs and birth defects and must be considered when interpreting the results of the first three studies; in fact, when such confounding is considered, there is no association between Zoloft and birth defects. Each Danish study will be reviewed in turn.

1. Wogelius

In 2006, Wogelius published a cohort study of Danish women from 4 Danish counties who gave birth from 1991 through 2003 (Wogelius P et al., Maternal use of selective serotonin reuptake inhibitors and risk of congenital malformation, *Epidemiology*, 2006 Nov;17(6):701-04).[9] The study did not examine individual SSRIs (including Zoloft) separately, but does

[9] The analyses adjusted only for smoking; maternal age; birth year; prescriptions for antiepileptics, antidiabetics, and NSAIDs; and county. Women who used SSRIs were more likely to be older, give birth in later years, come from different counties, smoke, have preterm births, and use prescriptions for NSAIDs and antiepileptics. The authors note that previous SSRI use (i.e., not during pregnancy) was also associated with a higher proportion of congenital malformations but they did not observe an association with non-SSRI antidepressants or with SSRI use late in pregnancy. However, they do not present the adjusted relative risk results for any of these comparisons.

highlight very clearly the problem of false positive results. Specifically, the authors reported a relative risk of 1.84 (95% CI of 1.25-2.71) for SSRI use in the 2nd or 3rd month after conception (a specific period within the first trimester) and overall congenital defects. However, as noted below in their follow-up study (Kornum 2010, discussed next), this association was wholly spurious.

2. Kornum

The analysis of this cohort was then updated in a paper by Kornum (Kornum JB et al., Use of selective serotonin-reuptake inhibitors during early pregnancy and risk of congenital malformations: Updated analysis, *Clin Epidemiol* 2010 Aug 9;2:29-36). In this paper, the authors used data from 1991 through 2007.[10] They present results from 28 comparisons. The ORs for septal heart defects among Zoloft users was 3.3 (95% CI of 1.5-7.5), based on only 6 exposed cases. The authors also restricted the analyses to those infants with cardiac malformations who also had heart surgery or catheter-based interventions and the OR was 2.3 (95% CI of 1.1-4.6), but they do not present results by specific SSRI nor do they present results separately for septal defects. It is important to note that the OR for SSRI use and overall malformations when examining SSRI use in the 2nd or 3rd month after conception was only 1.1 (95% CI: 0.8-1.6). This is not consistent with their prior study which reported an OR of 1.84 (95% CI: 1.25-2.71) for 2nd or 3rd month exposure, nor with their hypothesis that SSRIs would have a higher risk in these months of pregnancy if there were a true association. Thus,

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[10] 13 out of 17 years of data (76%) overlapped with Wogelius and many of the women included in this study were probably also included in the Pedersen study (discussed below). The analyses were adjusted only for smoking, birth order, maternal age, and birth year.

with a larger dataset, there is no significant association between SSRIs and overall malformations for this time period, demonstrating the unreliability of their prior analyses (likely a chance finding due to Type I error). The authors also report an OR for overall malformations of 1.1 (95% CI 0.8-1.4) for SSRI use in the period from six months to 30 days before conception (i.e., not during pregnancy), a result not different from use of SSRIs in the 2nd or 3rd month after conception (OR 1.1). Thus, these results do not take into account confounding by indication (and these results are better studied in the subsequent analyses in the Danish cohort, discussed below). In fact, the authors noted: "like most previous studies on this topic, we were unable to control for maternal disease type and severity" and, further, "we cannot exclude the possibility that increased risk of congenital malformations is caused by the disease underlying SSRI use or by other disease-related factors, rather than by SSRI use itself." In addition, the authors specifically noted the possibility of a detection bias (a potential bias that their study did not account for): "the mothers being treated for depression are more likely to receive or seek out more comprehensive prenatal and postnatal testing of their infants [which] may lead to more complete detection and registration of the less severe congenital malformations."

The study did not present any data for pre-pregnancy exposure and cardiac malformations, or any Zoloft-specific data for this comparison; these are critical omissions that diminish the utility of this study. In fact, the later Danish study by Jimenez-Solem demonstrates that the potential relationship between cardiac defects and SSRIs is the same in those exposed more than 30 days before conception or after delivery as those exposed in the first trimester, demonstrating that the findings identified in Kornum are most likely due to confounding by indication or detection bias. (Jimenez-Solem E et al., Exposure to selective serotonin reuptake

inhibitors and the risk of congenital malformations: a nationwide cohort study, *BMJ Open*, 2012 Jun 18;2(3))

3. Pedersen

In 2009, Pedersen published a study, using a nationwide registry of women in Denmark (Pedersen LH et al., Selective serotonin reuptake inhibitors in pregnancy and congenital malformations: Population based cohort study, *BMJ*, 2009 Sep 23;339:b3569). This cohort included children born from 1996 through 2003, and thus probably included many of the same subjects included in Wogelius 2006 and in Kornum 2010. The authors performed about 45 different comparisons.[11] They used filled prescriptions to identify SSRI use and, unlike prior and subsequent studies, required two prescriptions for SSRIs for the primary measure of exposure. There was no statistically significant relationship between Zoloft use and major malformations, major cardiac malformations, and major non-cardiac malformations. The study reported an odds ratio of 3.25 (95% CI of 1.21-8.75) for Zoloft and septal defects, based on only 4 exposed cases, which could be due to chance. As the authors noted: "The study was designed to investigate the potential association between SSRIs and several malformations with no adjustment for multi-hypotheses testing, and the findings could potentially have occurred by chance."

As discussed above, the Pedersen analysis is not independent of the Kornum 2010 analysis. In fact, Pedersen identified 4 septal heart defects in patients exposed to Zoloft in

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[11] They adjusted for only a few variables (age, calendar year, income, marriage status, and smoking); smoking information was missing in 17% of subjects. They did not adjust for other potential confounders such as alcohol use, vitamin use, opioid use, etc. They also did not account for the indication for the drug.

Denmark from 1996 through 2003 and Kornum identified 6 patients from 1991 through 2007; therefore, most of the 6 patients in the Kornum paper are likely the same patients as in Pedersen.

When examining any prescription as evidence for exposure to SSRIs, the finding for Zoloft was no longer statistically significant (OR 2.01, 95% CI: 0.83-4.86). The authors hypothesize that the use of two or more prescriptions results in more certainty of exposure to SSRIs, and thus the higher OR when using this definition. However, this could just as likely represent confounding by severity of disease. That is, requiring 2 or more prescriptions of SSRIs could simply weight the analysis towards the women with more severe depression who need to continue taking the drug during pregnancy (compared with women who are able to stop the drug during pregnancy), creating even stronger confounding.

Interestingly, the other Danish studies (Wogelius 2006 and Kornum 22010) which include Dr. Pedersen as a co-author, did not examine 2 or more SSRI prescriptions as a measure of exposure, and Pedersen did not state whether the use of 2 or more SSRI prescriptions as a measure of exposure was chosen before or after looking at the results. If the latter, it would double the number of comparisons made and increase the probability of a chance finding.

Like the Kornum study, Pedersen did not control for confounding by indication: "The disentanglement of the effects of treatment from the effect of the disease is a profound problem in pharmacoepidemiological studies."

4. Jimenez-Solem

A follow-up study using the Danish population was published by Jimenez-Solem in 2012 (Jimenez-Solem E et al., Exposure to selective serotonin reuptake inhibitors and the risk of congenital malformations: A nationwide cohort study, *BMJ Open*, 2012 Jun 18;2(3)) and included women from 1997 and 2009. This study was specifically designed to assess the potential for confounding by indication referenced in the earlier studies, which those predecessor

studies were not able to do. As the authors noted: "None of these [earlier] studies have successfully managed to differentiate between the consequences of the drugs themselves and the underlying disease." In order to do this, the study examined the relationship between SSRI use outside of the pregnancy period (but not during pregnancy) with birth defects. If SSRI use itself was responsible for birth defects, and not the indication for the drug or other factors that are associated with the use of SSRIs, then the use of SSRIs outside of the period of pregnancy should not be associated with birth defects. On the other hand, if there is an association between SSRI use during pregnancy and birth defects and if it is due to uncontrolled confounding (including confounding by indication), then one might expect to see a similar relationship between those who use SSRIs prior to or after pregnancy, but not during pregnancy, and birth defects. The study demonstrated the latter. Specifically, the study demonstrated that women who paused SSRI use during pregnancy (defined as use of SSRIs 3-12 months before conception and 1-12 months after giving birth but not between 3 months before to one month after giving birth) had an elevated risk of birth defects indistinguishable from that identified among women who used SSRIs during pregnancy. For example, with respect to atrial septal heart defects, the OR for first trimester SSRI exposure was 2.60 (95% CI: 1.84 to 3.68), the OR for first trimester Zoloft exposure was 2.85 (95% CI: 1.35-5.99), and the OR for paused SSRI use was 2.61 (95% CI: 1.17-5.84). The OR for women who did not use SSRIs but had an indication for them was thus the same as the OR for women who used SSRIs in the 1st trimester. The same is true if one compares the exposed and paused findings for other congenital heart malformations. As the authors noted, there was "nearly an identical risk for women who used an SSRI before and after pregnancy but discontinued use during pregnancy." The Jimenez-Solem study also examined various non-cardiac malformations, reporting no statistically significant increased risk between

Zoloft and the following malformations: nervous system; eye; ear, face, and neck; respiratory; orofacial clefts; digestive system; internal urinary system; external genital organs; limbs; and musculoskeletal.

In addition, the study reported no evidence of a dose-response relationship; all comparisons between high- and low-dose SSRIs were statistically insignificant.[12]

The study also could not account for detection bias (e.g., more screening in women on SSRIs) (Bar-Oz et al., Paroxetine and congenital malformation, *Clin Ther*, 2007 May;29(5):918-26). As the Jimenez-Solem noted: "Pregnant women exposed to SSRIs are reported to have increased rates of observed malformations due to increased rates of ultrasound examinations compared with women not treated with SSRIs." They went on to state: "More frequent echocardiograms could increase the risk of heart defect detection and give rise to information bias (diagnostic suspicion bias)."

5. Summary of Danish Studies

The Danish studies demonstrate that there is no drug-related association between Zoloft exposure and any congenital malformation. In particular, the Jimenez-Solem study highlights the importance of adequately controlling for biases and confounding, including confounding by indication, which earlier studies admittedly were unable to do. As the authors concluded: "risks related to SSRI use during the first trimester are a result of an unaccounted for confounder associated to the redemption of an SSRI prescription." Taken together, these four studies do not

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[12] No relationship was found between non-SSRI antidepressants, but this was based on only 223 tricyclic antidepressant users in one analysis and 831 users of other anti-depressants in another (with no data presented on the number of affected infants), compared with the 4,183 SSRI users.

provide evidence for a causal relationship between SSRIs generally, and Zoloft specifically, and birth defects.

F. Norwegian Population Studies

A study using data from the Norwegian Mother and Child Cohort Study (Nordeng H et al., Pregnancy outcome after exposure to antidepressants and the role of maternal depression, *J Clin Psychopharmacol*, 2012 Apr;32(2):186-94) examined four SSRIs individually (sertraline, paroxetine, citalopram/escitalopram, and fluoxetine) and as a group and attempted to isolate a drug-related effect from a woman's underlying depression or other confounders. As the authors noted, the date from earlier studies "strongly suggest that the various studies may suffer from uncontrolled and possibly unrecognized sources of bias." In an attempt to isolate a drug-related risk, the study examined three groups of women: (1) those women exposed to antidepressants during pregnancy; (2) those women who were exposed to antidepressants during the 6 months before pregnancy (called the "prior-only" group); and (3) those women who were not exposed to antidepressants in the 6 months before or during pregnancy.[13] By using these groups and controlling for additional factors, the investigators attempted to adjust "for maternal level of depression and a wide range of other potentially confounding factors." All women in the study also had a routine fetal ultrasound at weeks 17-18, mitigating concerns about detection bias. The study found no statistically significant increased risk of any malformation in children born to

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[13] Compared with non-users, both women with prior antidepressant use and with antidepressant use during pregnancy had less education; were less likely to have a normal BMI; and were more likely to smoke, to be hospitalized during pregnancy, to have asthma, and to take psychotropic drugs and analgesics. Of note, the presence of these factors was greater in general among women who used antidepressants during pregnancy than women who had prior use.

women who consumed Zoloft during pregnancy, OR 0.93 (95% CI: 0.34-2.53), with a similar point estimate compared with the prior-only group, OR 1.09 (95% CI: 0.74-1.62). The study also examined major malformations and cardiac malformations; however, there were so few cases reported for the Zoloft-exposed group that the authors could not calculate an odds ratio. The odds ratios for all SSRIs as a group in these categories were not statistically significantly increased: for all SSRIs, the odds ratio for major malformations was OR 1.07 (95% CI: 0.60-1.91), for cardiovascular malformations was OR 1.51 (95% CI: 0.67-3.43), and for septal defects was OR 1.57 (95% CI: 0.64-3.87). Based on these results, the investigators found "no increased risk of malformations after first-trimester exposure to antidepressants in general or SSRIs as a group," and "none of the individual SSRIs gave an overall increased risk of malformations."

G. Canadian Population Studies

A study published by Ramos et al. (including Dr. Anick Bérard) limited their cohort to women with at least one psychiatric disorder before pregnancy and use of antidepressants at least 30 days prior to pregnancy (Ramos E et al., Duration of antidepressant use during pregnancy and risk of major congenital malformations, *Br J Psychiatry*, 2008 May;192(5):344-50). In this way, the study attempted to account for confounding by indication. There was no statistically significantly increased risk of major congenital malformations with non-paroxetine SSRIs (OR 1.19, 95% CI: 0.71 1.97). In addition, there was no relationship between duration of antidepressant use and malformations. In fact, the OR decreased with increasing duration of exposure, a relationship opposite to that expected if there was a true biological relationship between antidepressants and birth defects. This study was limited by examining only major malformations as a single outcome and by limited data on individual SSRIs. Nonetheless, the study does provide evidence that certain findings between SSRIs and birth defects seen in other studies could be due to uncontrolled confounding by indication and not in fact true associations.

H. British Population Studies

In a study performed by the FDA, Margulis specifically tried to control for confounding more fully, including confounding by indication, through the use of propensity score matching on multiple factors (including diagnoses of depression, other mental health disorders, alcohol use, cigarette smoking, and index of multiple deprivation) (Margulis AV et al., Use of selective serotonin reuptake inhibitors in pregnancy and cardiac malformations, *Pharmacoepidemiol Drug Saf*, 2013 Sep;22(9):942-51). They used a UK electronic medical record database of over 11 million individuals that had information on these and other diagnoses and measures of health care utilization. Data on 149,464 births from 1996 through November 2010 were used. Women who used SSRIs had more comorbidities, less health encounters, and more other exposures compared with women who did not use SSRIs.[14] In adjusted analyses, there were no significant associations between SSRIs and any cardiac malformations, including septal defects, and all ORs were very close to one. This study is thus further support that SSRIs (and Zoloft) are not associated with cardiac malformations. As the authors noted: "The results of this study are most compatible with no association between maternal use of SSRIs in early pregnancy and cardiac malformations or septal defects in the offspring." The authors noted that the study "provides a further piece of evidence of the safety of SSRIs with regards to cardiac malformations."

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[14] They were more likely to smoke; have a diagnosis of depression; a diagnosis of other mental health conditions; fewer health care encounters; and use of antidepressants, non-antidepressant antipsychotics, anticonvulsants, and multiple other medication uses before pregnancy.

I. Summary of Studies That Have Attempted to Account for Indication for SSRIs

Several recent studies attempted to account for SSRI indication and thus underlying differences between women who use SSRIs and women who don't through various methods. What is most consistent about these studies is that none identified an association between SSRIs, and specifically Zoloft, and birth defects. These studies thus provide important results demonstrating the importance of accounting for confounding by indication and that in fact there is no true drug-related association between Zoloft and birth defects. To briefly summarize:

The Jimenez-Solem study, discussed above, specifically aimed to identify indication as a confounder. The study showed that women who used SSRIs before or after pregnancy (but not during) had the same relative risk as women who used SSRIs/Zoloft during the 1st trimester. For example, if one were to compare the risk of atrial septal defects among women who used SSRIs in the 1st trimester and compare it with the risk of atrial septal defects among women who used SSRIs only before and/or after pregnancy, the unadjusted OR would be 1.09 (P=0.9). If one were to do a similar calculation for Zoloft and atrial defects, comparing Zoloft exposed women in the 1st trimester with women who paused SSRIs during pregnancy, the unadjusted OR would be 1.3 (P=0.62). That is, the risk among women who used SSRIs (and Zoloft) is the same as the risk among women who paused SSRIs (i.e., had an indication for SSRIs). A paper written by Koren and Nordeng, based on the results of the Jimenez-Solem paper, noted: "These data strongly support the view that SSRIs do not increase the risk of cardiovascular malformations." (Koren G and Nordeng HM, Selective serotonin reuptake inhibitors and malformations: Case closed?, *Semin Fetal Neonatal Med*, 2013 Feb;18(1):19-22) The paper further stated: "SSRIs can probably be added to the infamous list of drugs wrongly incriminated as human teratogens,

only to be acquitted from being major teratogenic agents after much suffering by expecting mothers and their families."

A recent study of SSRIs as a group that controlled for confounding also reported that there was no association with cardiac malformations. The FDA study by Margulis directly adjusted for numerous confounders in their analysis, including an attempt to control for confounding by indication. With this adjustment, there were no significant associations between SSRIs and any cardiac malformations, including septal defects. The fact that most adjusted ORs were very close to one is also consistent with no effect of SSRIs after accounting for confounding.

Ramos attempted to account for confounding by indication by limiting their cohort to women with at least one psychiatric disorder and use of antidepressants before pregnancy. As noted, there was no statistically significantly increased risk of major congenital malformations with SSRIs, and there was, in fact, a duration response opposite to that expected if there was a true biological relationship between antidepressants and birth defects.

J. Overall Summary of Findings of the Individual Studies

In summary, the extensive research done to date has not demonstrated a relationship between Zoloft and congenital abnormalities. Despite design limitations of the studies that would tend to bias them toward reporting associations when no true association was in fact present, there is an extensive body of data examining Zoloft specifically and individual malformations, which has not demonstrated any relationship. There is also additional strong evidence for confounding by risk factors and by indication, for differential ascertainment, and for Type I error. All of these factors could create false positive associations, rather than true associations.

Considering the data as a whole, there is no evidence that Zoloft increases the risk of any malformations, including major cardiac malformations and septal defects.

K. Meta-Analysis

Meta-analysis is a formal statistical method of grouping data together that can enhance power and allow for examination of differences among studies. Because a meta-analysis of observational studies combines data from the underlying studies, such a meta-analysis suffers from the same inherent biases that apply in the individual observational studies being analyzed, which in the case of SSRIs (and Zoloft) tends to bias the meta-analysis in the same way that the individual studies are biased. That is, a meta-analysis of the studies described above would tend to report associations due to chance, confounding, recall bias, and ascertainment bias – i.e., to report associations that are not due to the drug under study but rather other factors. One must assess the validity of individual meta-analyses, just as one assesses the validity of individual studies. Despite these factors tending to bias results toward reporting an association when no true association exists, the only meta-analysis that, to my knowledge, examined Zoloft specifically found that there was no statistically significant increased risk of major malformations or cardiac malformations. (Myles N et al., Systematic meta-analysis of individual selective serotonin reuptake inhibitor medications and congenital malformations, *Aust N Z J Psychiatry*, 2013 Jun 12) In addition, the ORs were all very close to one, with narrow confidence intervals. Furthermore, the results were consistent with a lack of a class effect.

L. Studies Examining the Relationship Between SSRIs and Persistent Pulmonary Hypertension (PPHN) of the Newborn, Focusing on Zoloft

A number of studies have examined whether there is an association between SSRI use and persistent pulmonary hypertension of the newborn (PPHN). These studies are discussed in turn.

1. Chambers

A study by Chambers was, to my knowledge, the first published study examining the possible relationship between SSRIs and PPHN (Chambers CD et al., Selective serotonin-reuptake inhibitors and risk of persistent pulmonary hypertension of the newborn, *N Engl J Med*, 2006 Feb 9;354(6):579-87). This study used a case-control design of U.S. patients and relied on interviews with women after delivery to identify medication exposure, subjecting the study to recall bias. In addition, participation rates were only 68-69%. The study did not present results for individual SSRIs such as Zoloft. When examining all SSRIs as a group, the odds ratio for SSRI use before week 20 was 0.3 (95% CI: 0.1 1.2) and after week 20 was 6.1 (95% CI: 2.2-16.8). The study, however, did not adjust for several important confounders, including sex of the baby (which was significantly associated with PPHN in this study), several risk factors that the authors identified as potential risk factors in the same patient population (cesarean section, maternal asthma, birth weight) (Hernandez-Diaz S et al., Risk factors for persistent pulmonary hypertension of the newborn, *Pediatrics*, 2007 Aug;120(2):e272-82) (Chung TK et al., Antepartum depressive symptomatology is associated with adverse obstetric and neonatal outcomes, *Psychosom Med*, 2001 Sep;63(5):830-34), indication for the drugs, and pregnancy complications. Thus, the study is limited by its design and does not provide data demonstrating any increased risk for Zoloft specifically.

2. Källén

A study by Källén using Swedish data used the same design as described previously for birth defects (Källén B and Olausson PO, Maternal use of selective serotonin re-uptake inhibitors and persistent pulmonary hypertension of the newborn, *Pharmacoepidemiol Drug Saf*, 2008 Aug;17(8):801-06). In particular, exposure to SSRIs was ascertained from most women during the first trimester. Although this minimizes recall bias, it does not provide data on the primary

exposure of interest for PPHN: exposure during the third trimester. The authors therefore relied on prescription data for this exposure among women with infants with PPHN and only examined this among those exposed in the first trimester. That is, they did not appear to try to ascertain all exposures in the population after the 1st trimester, but only in those women with infants with PPHN who reported use in the first trimester. This would underestimate use of SSRIs among women with infants without PPHN, which would create a spurious association. The study also does not provide ORs specifically for Zoloft and, of the three women reporting Zoloft use in the first trimester, only one had a documented prescription for Zoloft later in pregnancy. Thus, this study does not provide any data demonstrating a relationship between Zoloft and PPHN. Moreover, a follow up, and much larger, study which included data from Källén (the Keiler study, discussed below) demonstrated that the finding for SSRIs as a group was most likely due to confounding.

3. Andrade

Another study, by Andrade et al. (Andrade SE et al., Antidepressant medication use and risk of persistent pulmonary hypertension of the newborn, *Pharmacoepidemiol Drug Saf*, 2009 Mar;18(3):246-52), used data from four health plans in the U.S. This study compared 1,104 women who received antidepressants in the 3rd trimester with 1,104 matched controls (women who did not receive antidepressants). The study reported no statistically significant difference between SSRI users and non-users, albeit with wide confidence intervals (prevalence ratio of 0.79 with 95% CI of 0.07-6.89). This study was limited by its small size and it did not examine SSRIs individually.

4. Wilson

More recently, a study examining PPHN in women treated at the Madigan Army Medical Center by Wilson was published (Wilson KL et al., Persistent pulmonary hypertension of the

newborn is associated with mode of delivery and not with maternal use of selective serotonin reuptake inhibitors, *Am J Perinatol*, 2011 Jan;28(1):19-24). Cases of PPHN were compared with a sample of controls without PPHN. Medication use was measured using both filled prescriptions and documentation in the medical records. The study examined various potential risk factors for PPHN including tobacco use, infant gender, mode of delivery, maternal disease, BMI, and maternal age, and confirmed the infant's PPHN diagnoses through echocardiogram. The study found that there was no statistically significant association between SSRIs as a group and PPHN (data were not reported for Zoloft individually). In addition, none of the 20 cases of PPHN were exposed to SSRIs after 20 weeks of gestation, which is inconsistent with an association between SSRIs and PPHN. Interestingly, however, the study did report an association between mode of delivery and PPHN – cesarean births had a 4.9-fold higher odds ratio. This study suggests that the earlier studies failed to control for important confounders (e.g., mode of delivery or pregnancy complications that may lead to cesarean sections). This study was limited by its small size and lack of Zoloft-specific data. Nonetheless, it found no association between SSRIs as a group and PPHN.

5. Kieler

Kieler subsequently published, to my knowledge, the largest study to date using data from Denmark, Iceland, Finland, Norway, and Sweden (Kieler H et al., Selective serotonin reuptake inhibitors during pregnancy and risk of persistent pulmonary hypertension in the newborn: Population based cohort study from the five Nordic countries, *BMJ*, 2012 Jan 12;344:d8012). PPHN cases from Källén 2008 were included in this study, although the degree of overlap was not specified. The authors identified almost 2,000 cases of PPHN, almost 4 times as many as the largest previous study (Källén 2008) and over 5 times more than Chambers 2006. Unlike other studies, they also assessed for the possibility of confounding by indication by

analyzing both women who only used SSRIs up through the first 8 weeks of pregnancy and women with prior psychiatric admissions who did not use SSRIs. In addition, they adjusted for some but not all potential confounders (e.g., they did not adjust for maternal asthma, illicit drug use, and pregnancy complications other than meconium aspiration). The study reported risks for PPHN in users of SSRIs at or later than 20 weeks of pregnancy that were the same as those between women using SSRIs only in early pregnancy and PPHN and between prior psychiatric admissions and PPHN in the absence of SSRI use. Thus, these findings (like the studies on congenital malformations) provide evidence of confounding by indication. More specifically with respect to Zoloft, if one compares use of Zoloft at or later than 20 weeks (10 PPHN and 2833 non-PPHN subjects) with Zoloft use only in the first 8 weeks of pregnancy (9 PPHN and 3,387 non-PPHN subjects), the crude relative risk is 1.37 (95% CI: 0.54 3.26, P=0.54). That is, there was no statistically significant difference in the risk of PPHN among women exposed to SSRIs during the time period hypothesized to be of biological importance and women not exposed during this time period but who had an indication for SSRIs.

In summary, the most recent, largest study is consistent with no true association with PPHN.

M. Overall Summary of Evidence

1. There Is No Evidence for a Class Effect Among SSRIs

As noted above, a class effect of teratogenicity for a group of drugs cannot be assumed (Mitchell 2012). There are numerous pieces of evidence supporting a lack of class effect among SSRIs and birth defects. First, there is objective evidence of a different effect of SSRIs on birth defects, particularly cardiac defects. (Myles 2013) (Reis & Källén 2010) (Källén 2006). Specifically, statistical tests for difference among SSRIs demonstrate that the effects of SSRIs are not uniform. Second, if there truly were a class effect of SSRIs, then the effect of all SSRIs

combined should be similar to the effect of individual SSRIs. For example, if all SSRIs truly increased the risk of cardiovascular defects two-fold, then the odds ratio for all SSRIs combined and cardiovascular defects should be two, as should the odds ratio for the individual SSRIs. This is not the case across the literature. A single elevated odds ratio for one SSRI when there is no overall increase in risk from SSRIs is thus inconsistent with a class effect. Thus, there is evidence against a class effect for SSRIs and one must therefore examine the effect of Zoloft separately.

Consistent with this conclusion, only paroxetine has been deemed to be a Category D drug by the FDA, while all other SSRIs including Zoloft are in Category C. This is also consistent with statements by commentators who have reviewed the literature. For example, one commentator noted, a "high degree of heterogeneity among such results as been identified. This heterogeneity is detectable both in research investigating SSRIs as a group and in research more correctly focused on investigating the teratogenic potential of single SSRI agents." (Gentile S, Selective serotonin reuptake inhibitor exposure during early pregnancy and the risk of birth defects, *Acta Psychiatr Scand*, 2011 Apr;123(4):266-75).

However, even if one were to examine the results for all SSRIs as a group (not just Zoloft), there is still no evidence of a drug-related increased risk of birth defects. As noted previously, in conducting my analysis and considering the arguments made by plaintiffs' experts, I considered studies of all SSRIs (not differentiated by drug). Although these data includes paroxetine (a Category D drug), the overall data from the studies supports my opinion that there is no causal association with birth defects. A number of the studies above that report data for Zoloft also report data for SSRIs as a group, as well as numerous studies that have examined SSRIs overall (for example Vasilakis-Scaramozza C et al., Antidepressant use during early

pregnancy and the risk of congenital anomalies, *Pharmacotherapy*, 2013 Jul; 33(7):693-700), show no association with birth defects. Moreover, as noted above, recent studies that have attempted to control for confounding by indication have uniformly found no increased risk.

2. Zoloft Is Not Associated With an Increased Risk of Any Birth Defects

There are numerous pieces of evidence that demonstrate a lack of association between Zoloft and birth defects.

First, there is a large body of evidence examining Zoloft specifically, which finds no association for the majority of malformations studied, including major cardiac malformations.

Second, as noted previously, the play of chance can cause spurious associations. It is not surprising that results from one study are not reproduced in other studies because these are not true associations; they are simply Type I error.

Third, along with the play of chance (Type I error), it is very likely that studies examining Zoloft and birth defects contain inherent biases including: (1) *Confounding by indication*. Several studies have demonstrated how confounding by indication can cause spurious associations (see above). Early studies failed to control for this confounder. Later studies which have best been able to control for underlying disease have demonstrated no associations between Zoloft and birth defects. (2) *Other confounding*. Women who use SSRIs are clearly different in many ways from women who do not use SSRIs, and studies that have identified positive findings have failed to adjust for these important differences. It has been clearly demonstrated that confounding can cause spurious associations between SSRIs and birth defects, not true associations (Diav-Citrin 2008) (Malm 2011) (Bérard abstract 2009). (3) *Differential ascertainment*. Women who use SSRIs and their infants are more likely to undergo screening tests that might reveal birth defects that would otherwise go unnoticed. In addition, infants born to mothers who use SSRIs are more likely to have non-birth defect related

perinatal findings that would lead to more scrutiny and identification of birth defects that might otherwise go unnoticed. These latter two points are particularly relevant for birth defects such as ASDs and VSDs and demonstrate why findings for such malformations should be interpreted with caution.

Fourth, the meta-analysis by Myles 2013 demonstrates no association between Zoloft and major or cardiac malformations, with odds ratios of one or lower and narrow confidence intervals.

Thus, the available evidence does not demonstrate an association between Zoloft and any congenital defects.

3. There Is No Evidence for a Causal Association Between Zoloft and PPHN

As discussed above, there is no evidence of a causal association between Zoloft and PPHN. The earlier studies of PPHN failed to control for recall bias, non-participation bias, and confounding and did not specifically examine Zoloft. Furthermore, the study by Kieler (2012) demonstrates that there is no true association between Zoloft use and PPHN when one attempts to control for factors associated with SSRI use or the underlying psychiatric disorders.

4. The General Scientific Consensus Is that Zoloft Has Not Been Shown to Cause Birth Defects or PPHN

Given the evidence presented above, it is not surprising that other scientists and scientific and regulatory groups have concluded that a causal relationship has not been demonstrated between SSRIs (and specifically Zoloft) and birth defects or PPHN (Koren 2013) (Myles 2013) (Margulis 2013) (FDA Drug Safety Communication 2011) (Canadian Paediatric Society Position Statement 2011) (Yonkers 2009 (ACOG)). For example, the authors of the recent FDA study on birth defects concluded: "The results of this study are most compatible with no association between maternal use of SSRIs in early pregnancy and cardiac malformations or septal defects in

the offspring" (Margulis 2013). In addition, as noted above, the FDA has placed Zoloft in pregnancy Category C, which means there are no well-controlled studies demonstrating an increased risk in humans. This is consistent with statements from scientific reviewers. For example, a review by Koren (2013) concluded: "SSRIs can probably be added to the infamous list of drugs wrongly incriminated as human teratogens, only to be acquitted from being major teratogenic agents after much suffering by expecting mothers and their families." A review by Lorenzo similarly stated: "In summary, there is no evidence that sertraline increases the overall risk for major malformations." (Lorenzo L, Antidepressant use in pregnancy, *Expert Opin Drug Saf*, 2011 Nov;10(6):883-89)

The FDA has made similar statements regarding the lack of a causal association with PPHN. In a recent statement concerning PPHN, the FDA stated that "[a]t present, FDA does not find sufficient evidence to conclude that SSRI use in pregnancy causes PPHN, and therefore recommends that health care providers treat depression during pregnancy as clinically appropriate" (FDA, Drug Safety Communication, 2011). Similarly, in conjunction with revisions to the Zoloft labeling, the FDA noted: "At present we find insufficient evidence to demonstrate a causal association between SSRI use in pregnancy and PPHN." (FDA Letter to Pfizer, 2011 Jun 29)

VII. Plaintiffs' Expert Reports

I have reviewed the reports of plaintiffs' experts, particularly those of Dr. Bérard and Dr. Vekemans which address epidemiologic issues. Overall, there are numerous methodological flaws and limitations to Dr. Bérard's and Dr. Vekeman's approach, some of which have been highlighted very briefly at the beginning of this document. I will review the individual findings of these experts below.

A. Dr. Bérard

Although earlier in her report Dr. Bérard reviews the study designs and points out some of the design characteristics and limitations of those studies, she presents selected results from those studies later in her report without considering the reliability of those results nor presenting the vast amount of data that contradict her conclusions. Her approach thus does not account for study design biases and limitations, the lack of consistency in the literature, nor the strong likelihood of Type I errors. Dr. Bérard also often relies on data from SSRIs other than Zoloft, without noting the considerable evidence (discussed above) against a class effect.

More specifically, Dr. Bérard presents only selected relative risks and odds ratios from various studies and does not present the vast amount of contradictory data. One should not assume that there is an association and then identify only data that are consistent with this assumption. As one example, she presents results on SSRI exposure in months 2 and 3 of pregnancy from Wogelius' 2006 study but fails to present the results from the follow-up to that study, using the same but larger population (Keiler 2012), that refuted those findings.

Dr. Bérard claims there is evidence of a dose-response relationship, citing Jimenez-Solem, but that study found no such relationship. And she ignores her own study which found no evidence of a duration-response relationship (Ramos 2008).

Dr. Bérard fails to cite other studies altogether. Thus, for example, she does not cite Myles 2013, the recent meta-analysis that analyzed Zoloft separately. Instead, she cites an earlier analysis by Nikfar that does not report data for Zoloft, but rather only for all SSRIs as a group, and includes overlapping patient populations (i.e., it double counts patients) and excludes numerous published studies. (Moreover, when Dr. Bérard cites Nikfar, she does so selectively and overlooks the fact that the paper reports no association with cardiac and minor malformation.) (Nikfar S et al., Increasing the risk of spontaneous abortion and major

malformations in newborns following use of serotonin reuptake inhibitors during pregnancy, *Daru*, 2012 Nov 1;20(1):75)

Similarly, while Dr. Bérard takes the position that SSRIs should be examined as a "class," she ignores the FDA's recent study finding that there is no association between SSRIs as a group and cardiac malformations. And she ignores that her own study found no increased risk for birth defects for SSRIs as a group (excluding Paxil). (Ramos 2008). In sum, Dr. Bérard ignores the large body of epidemiologic data finding no association between Zoloft and birth defects as well as a large body of data finding no association between all SSRIs as a group and birth defects.

In her approach, Dr. Bérard also relies on many results that are not statistically significant and essentially ignores statistical significance and CIs. However, the standard statistical approach is to test the null hypothesis. That is, one assumes that there is no association and then tests to see if the results obtained are consistent with the null. As examples, Dr. Bérard notes that the OR for septal defects among all SSRIs users in Louik 2007 is 1.2 (95% CI of 0.8-1.8) as support that SSRIs as a class increase the risk of cardiac defects despite the fact that the results are not statistically significant; that is, the findings do not refute the null hypothesis of a lack of effect. One cannot assume that the OR of 1.2 in this example is correct nor that the results would be statistically significant if one could just study enough patients; the result itself may be incorrect due to inherent biases in the study, as previously noted. If one ignores this important fact, then, in the same study, one could conclude that SSRIs protect against craniosynostosis: the OR for craniosynostosis is 0.8 (95% CI of 0.2-3.5), which represent a 20% lower odds of this defect from SSRIs. Of course, neither of these findings is statistically significant. As another example, the ORs (and 95% CIs) for Zoloft and any congenital malformations and cardiac

malformations in Källén are 0.78 (95% CI: 0.61-1.00) and 0.76 (95% CI: 0.47-1.23), respectively. Using Dr. Bérard's approach, this study demonstrates a protective effect of Zoloft and should be used as evidence for benefit of the drug.

Equally importantly, Dr. Bérard ignores that the actual measures of effect from the studies she cites (odds ratios and relative risks) are not accurate representations of the true effect in the face of the many biases noted above (e.g., uncontrolled confounding, detection bias, recall bias, etc.). As one specific example, Dr. Bérard notes that Jimenez-Solem 2012 reported an OR for septal defects among women exposed in the first trimester of 2.04 as evidence of risk, but fails to note that the OR for septal defects among women who paused SSRIs during pregnancy was 2.56, a finding consistent with confounding by indication and overall no true association between SSRIs and septal defects (as discussed in detail above). Thus, Dr. Bérard does not account for the fact that the Jimenez-Solem study is evidence that there is not a true association between SSRIs and birth defects.

Even if an individual OR is statistically significant, one must consider the strong likelihood that findings are due to chance given the hundreds and hundreds of statistical tests being performed. This basic principle of epidemiology has been noted frequently by authors in the field. Dr. Bérard also presents results from multiple studies that are drawn from the same underlying population and thus do not provide independent confirmatory results.

Finally, Dr. Bérard suggests that there is an increased risk of spontaneous abortions associated with Zoloft and that this could have diminished a true association between Zoloft and birth defects. However, as noted earlier, many birth defects typically do not lead to spontaneous abortions including isolated VSDs and ASDs, many major cardiac malformations, and various non-cardiac malformations (e.g., club foot, craniosynostosis). In addition, several studies have

included at least some pregnancy terminations but have not reported increased risks of birth defects, including major cardiac malformations (Malm 2005) (Alwan 2007), further mitigating concerns of bias from these events. Further, a recent large study demonstrated no association between Zoloft and spontaneous abortions after accounting for underlying depression (Kjaersgaard M et al., Prenatal antidepressant exposure and risk of spontaneous abortion, *PLoS*, 2013 Aug 28:8(8):e72095). In addition, another recent study found no statistically significant increased risk of still birth associated with Zoloft. (Jimenez-Solem 2013) Thus, it is unlikely that studies of birth defects are biased by spontaneous abortions.

B. Dr. Vekemans

Dr. Vekemans similarly presents an incomplete review of the evidence. He does not critically evaluate the literature, including the potential for confounding, biases, and Type I error, as discussed above.

He also presents only data that he claims support his conclusions while ignoring the large amount of contrary data. For example, he discusses the results of Wogelius 2006, noting that the relative risk was 1.84 in women exposed to SSRIs during the 2nd or 3rd months of pregnancy, but fails to mention the follow-up study in a larger population demonstrating no risk of SSRIs during the 2nd or 3rd months of pregnancy (odds ratio 1.1). He also chooses to present selected results out of hundreds of comparisons made across studies, without discussing the likelihood of Type I error.

Dr. Vekemans also discusses the number needed to harm and attributable risk. However, these calculations are only meaningful if one has established that the association between SSRI and birth defects is both true and accurate. As discussed above, neither of these have been established. He also does not provide the underlying data used for these calculations.

In addition, while he double counts findings based on a similar data source (e.g., Pedersen 2009 and Kornum 2010), he ignores a basic principle of epidemiology that such publications cannot verify their own results.

He also does not discuss the important findings of Jimenez-Solem, Margulis 2013, and Ramos 2008, demonstrating the important role of confounding in many of the studies he cites and that there is no drug-related increased risk of birth defects when such confounding is taken into account. He discussed SSRIs as a class, without noting the considerable evidence (discussed above) against a class effect. He also discusses how a dose-response may be an important element in assessing causality, but ignores that studies have found no dose-response or duration relationship. (Jimenez-Solem) (Ramos 2008)

C. The Claims of Specific Associations Contained in Plaintiffs' Expert Reports Are Not Supported by the Data

Beginning on page 19 of her report, Dr. Bérard presents selected data from studies on SSRIs and birth defects and PPHN. Similarly, Dr. Vekemans presents only selected information and does not provide complete information in his Appendix A. I reviewed the various malformations that these experts discuss in their reports, which demonstrate that there is no evidence of an association between Zoloft and any malformation.

It is important to note that while Dr. Bérard and Dr. Vekemans group together various malformations, scientists in the field recognize that there are important differences between birth defects, meaning that they must be considered individually. For example, a study by Bakker noted: "Heart defects, as a group, are heterogeneous: the development of the heart is a complex process and a wide variety of heart defects can occur…. A specific exposure is not expected to increase the risk for congenital heart defects in general. In studying risk factors, it is therefore important to create homogeneous groups." (Bakker MK et al., First-trimester use of Paroxetine

and congenital heart defects, *Birth Defects Res A Clin Mol Teratol*, 2010 Feb;88(2):94-100) Nonetheless, because Dr. Bérard and Dr. Vekemans group together malformations in their reports, I will consider the individual malformations within each of these overarching categories created by plaintiffs' experts below.

a. Zoloft and All Major Malformations

The epidemiological evidence as a whole shows no association between Zoloft use and all major malformations as a group. Dr. Bérard fails to consider multiple studies that reported no statistically significant association for Zoloft and all major malformations: Reis & Källén 2010: OR 0.99 (95% CI: 0.81-1.21); Malm 2011: OR 1.00 (95% CI: 0.71-1.39); Colvin 2011: OR 1.01 (95% CI: 0.72-1.40); Alwan 2007: OR 0.9 (95% CI: 0.6-1.4); and Myles 2013: OR 1.01 (95% CI: 0.88–1.17). Dr. Bérard ignores these data, instead citing a selected three studies that do not support a statistically significant association. The first, Louik 2007, did not report the odds ratio included in Dr. Berard's report. The other two odds ratios that she presents from Kornum 2010 and Pedersen 2009 are not statistically significant and use overlapping data from Denmark (and thus are not independent of each other). She also does not consider the methodological limitations nor the evidence for confounding in Jimenez-Solem. Thus, given these data and all of the other considerations noted in my report previously, there is no evidence of an association between Zoloft and any malformations considered as a group.

b. Zoloft and Cardiac Malformations

The epidemiological evidence as a whole shows no association between Zoloft use and all cardiac malformations as a group. Dr. Bérard fails to consider several studies finding no association: Alwan 2007: OR 0.7 (95% CI: 0.4-1.3); Louik 2007: OR 1.5 (95% CI: 0.9-2.6); Reis & Källén 2010: OR 0.74 (95% CI: 0.50-1.09); Malm 2011: OR 0.65 (95% CI: 0.34-1.25); Colvin 2011: OR 1.74 (95% CI: 0.96-3.17); and Myles 2013: OR 0.93 (95% CI: 0.70-1.24).

Dr. Bérard instead presents the results of three Danish studies with overlapping data (Kornum 2010, Pedersen 2009, and Jimenez-Solem 2012) as though they are independent of each other (which they are not, as I note above), and she ignores the most important finding of Jimenez-Solem: the similar findings among the group of women who paused SSRIs during pregnancy. Thus, Jimenez-Solem in fact provides strong evidence against a true association. In addition, the OR she reports from Louik appears incorrect. She also reports an OR of 2.73 (95% CI: 1.75-4.26) for cardiac malformations, but the actual results from Louik do not show a statistically significant increased risk (OR 1.5 (95% CI: 0.9-2.6)), further support for my conclusion that there is no evidence demonstrating an association between Zoloft use and cardiac malformations as a group.

Dr. Bérard discusses various individual cardiac malformations as supposed support for her opinion about cardiac malformations. However, there is no evidence demonstrating a statistically significant association with the individual cardiac malformations discussed in her report:

(1) Zoloft and Conotruncal Heart Defects

The studies that have examined conotruncal defects have found that there is no association with maternal Zoloft use: Alwan 2007: OR 1.3 (95% CI: 0.6-2.7); Louik 2007: OR 0.7 (95% CI: 0.2-3.3); and Malm 2011: OR 1.27 (95% CI: 0.18-9.15). There is no study that has reported a statistically significant association between Zoloft and conotruncal heart defects. Dr. Bérard ignores these data.

(2) Zoloft and Transposition of the Great Arteries

Dr. Bérard ignores that the one study that examined transposition of the great arteries found no statistically significant increased risk with Zoloft: Malm 2011: OR 2.55 (95% CI:

0.35-18.62). There is no study that has reported a statistically significant association between Zoloft and transposition of the great arteries.

(3) Zoloft and Right Ventricular Outflow Tract

The three studies that examined right ventricular outflow tract defects found that there was no statistically significant association with Zoloft: Alwan 2007: OR 0.8 (95% CI: 0.3-2.3); Louik 2007: OR 2.0 (95% CI: 0.6-6.8); and Malm 2011, which reported no cases in the exposed group. Dr. Bérard ignores these findings. She also ignores that no study has reported a statistically significant association between Zoloft and right ventricular outflow tract defects.

(4) Zoloft and Left Ventricular Outflow Tract

The three studies that examined left ventricular outflow tract defects found that there was no statistically significant association with Zoloft: Alwan 2007: OR 0.4 (95% CI: 0.1-1.6); Louik 2007: OR 1.9 (95% CI: 0.6-5.8); and Malm 2011, which reported no cases in the exposed group. Dr. Bérard again ignores these studies. No study has reported a statistically significant association between Zoloft and left ventricular outflow tract defects.

(5) Zoloft and Anomalies of the Pulmonary Valve

One study has examined anomalies of the pulmonary valve and found no statistically significant increased risk with Zoloft: Colvin 2011, OR 2.20 (95% CI: 0.54-8.92). There is no study that has reported a statistically significant association between Zoloft and anomalies of the pulmonary valve. Dr. Bérard ignores this fact.

(6) Hypoplastic Left Heart Syndrome

Dr. Bérard ignores that the one study that has examined hypoplastic left heart syndrome found no statistically significant increased risk with Zoloft: Colvin 2011, OR 3.36 (95% CI: 0.82-13.78). There is no study that has reported a statistically significant association between Zoloft and hypoplastic left heart syndrome.

(7) Septal Defects (Combined)

There is no evidence that Zoloft increases the risk of septal defects combined. Dr. Bérard ignores the studies from multiple populations finding no statistically significant increased risk of septal defects and Zoloft: Alwan 2007: OR 0.7 (95% CI: 0.3-1.5); Colvin 2011: OR 1.13 (95% CI: 0.42-3.03); and Reis & Källén 2010: "No increased risk was seen for VSD and / or ASD with any of the three other SSRIs." Dr. Bérard instead cites a finding from the Louik 2007 study, which was not replicated in the companion study reported in the same edition of the *New England Journal of Medicine* (Alwan 2007). She also cites data from the Danish population studies, but ignores the results of Jimenez-Solem, which are most consistent with an absence of a drug-related increased risk of septal malformations with Zoloft use. Considering these data together, the available evidence does not support an association between Zoloft and septal defects combined.

(8) Ventricular Septal Defects

When examining specific septal defects separately as one should, the data demonstrate that there is no statistically significant association between Zoloft and ventricular septal defects. Dr. Bérard ignores the studies that have shown no association: Malm 2011: OR 0.53 (95% CI: 0.22-1.29) and Colvin 2011: OR 0.49 (95% CI: 0.07-3.50). The only data on which she relies is from the Jimenez-Solem 2012 study but, as I note above, this study supports a lack of a drug-related association with Zoloft – a finding that Dr. Bérard ignores.

(9) Atrial Septal Defects

Similarly, the data demonstrate that there is no statistically significant association between Zoloft and atrial septal defects. Dr. Bérard ignores the following studies, which found no association with atrial septal defects: Malm 2011: OR 0.93 (95% CI: 0.23-3.76) and Colvin

2011: OR 2.32 (95% CI: 0.57-9.42). Again, the only study Dr. Bérard cites is the Jimenez-Solem study; but, again, this study refutes, rather than supports, a true association.

c. Zoloft and Craniosynostosis

The epidemiologic evidence demonstrates no association between Zoloft and craniosynostosis. Dr. Bérard cites only one piece of data for Zoloft and craniosynostosis, that from Louik 2007 with an OR of 1.8 but a 95% CI of 0.2-14.9. This OR was based on only 1 exposed case and has very wide CIs, clearly not evidence for a true association. In her evaluation of any SSRIs and craniosynostosis, she also fails to note that the overall OR for SSRIs and craniosynostosis in Louik was 0.8 (95% CI of 0.2-3.5) and that Louik specifically states that "Our analysis did not confirm previously reported [Alwan 2007] associations between overall use of SSRIs and craniosynostosis.…" Dr. Bérard also fails to consider the additional multiple studies that found no increased risk between Zoloft and craniosynostosis (and failed to replicate the Louik finding): Alwan 2007: OR 1.7 (95% CI: 0.7-4.2) and Malm 2011: OR 2.42 (95% CI: 0.33-17.60). Thus, given these data and all of the other considerations noted in my report previously, the available evidence does not demonstrate an association between Zoloft and craniosynostosis.

d. Zoloft and Pulmonary/Respiratory Defects

Dr. Bérard discusses pulmonary/respiratory defects together in one section of her report. As an initial matter, Dr. Bérard ignores that respiratory defects and PPHN are distinct injuries; findings regarding PPHN cannot be used to demonstrate an association with respiratory malformations. Nonetheless, I consider both respiratory defects and PPHN in this section so as to respond to the data on which Dr. Bérard relies.

The epidemiological evidence demonstrates that there is no association between Zoloft use and respiratory defects. Dr. Bérard cites the finding from the Colvin 2011 study, but ignores

that this has never been replicated, as demonstrated by the Malm study: OR 0.69 (95% CI: 0.10-4.93) (Malm 2011). With respect to PPHN, she does not discuss the methodological limitations of these studies (as noted above) nor that one of the studies she cites (Kieler 2012) provides strong evidence for confounding by indication, rather than a true association.

Thus, the available evidence does not support an association between Zoloft and respiratory congenital birth defects. In addition, a causal association between Zoloft and PPHN has not been demonstrated as I noted above.

e. Zoloft and Gastrointestinal Defects

Dr. Bérard discusses numerous individual malformations under the umbrella of "gastrointestinal defects." Again, this grouping includes heterogeneous malformations; findings for one specific malformation should not be seen as evidence for an association for a different malformation. Nevertheless, the epidemiological evidence demonstrates that there is no association between Zoloft and any of the malformations that Dr. Bérard discusses in this grouping.

(1) Omphalocele

Recent studies from two study populations found no statistically significant increased risk of omphalocele in infants born to women who consumed Zoloft: Alwan 2007, OR 1.5 (95% CI: 0.4-6.6) and Malm 2011, which reported no cases of omphalocele in the exposed group. Dr. Bérard ignores these findings. She instead cites an odds ratio from the Louik 2007 study, which has never been replicated (including in the companion Alwan 2007 study), was based only on three cases, and was most likely a chance finding. She also cites data from Louik for all SSRIs combined, but ignores that the study authors stated that their study did not establish an association between SSRIs and omphalocele. Reviewing the data as a whole, there is no association between Zoloft and omphalocele.

(2) Gastroschisis

The one study that examined gastroschisis found that there was no increased risk in the Zoloft-exposed group: Alwan 2007, OR 0.9 (95% CI: 0.3-3.3). Dr. Bérard ignores this finding.

(3) Digestive system and Overall Gastrointestinal Malformations

The epidemiological evidence shows that there is no association between digestive system malformations and maternal Zoloft use. Studies have reported no statistically significant association: Malm 2011: OR 1.09 (95% CI: 0.35-3.41) and Jimenez-Solem 2012: OR 1.43 (95% CI: 0.36-5.74). Relatedly, the epidemiological evidence shows that there is no association between gastrointestinal malformations and maternal Zoloft use: Colvin 2011, OR 0.97 (95% CI: 0.40-2.36). Dr. Bérard ignores these data.

f. Anal and Anorectal Atresia

There is no evidence of an association between Zoloft and anal atresia or anorectal atresia. Dr. Bérard ignores that the Alwan study found no association between anorectal atresia and maternal Zoloft use: Alwan 2007, OR 0.7 (95% CI: 0.2-2.8). She instead cites a finding from Louik 2007, which has never been replicated, was based on three exposed cases, and was likely due to chance. Taken together, the evidence does not support an association between Zoloft and anal atresia or anorectal atresia.

g. Pyloric Stenosis

The one study that examined pyloric stenosis found no statistically significant increased risk after maternal exposure to Zoloft: Louik 2007, OR 1.7 (95% CI: 0.7-4.1). No study has reported a statistically significant increased risk for pyloric stenosis in infants born to women exposed to Zoloft during pregnancy.

h. Zoloft and Neural Tube Defects

Dr. Bérard discusses neural tube defects as a group; this includes both anencephaly and spina bifida. The data show that there is no increased risk of either malformation considered individually or for the malformations as a group for those exposed to Zoloft:

(1) Neural Tube Defects (Group)

Studies from multiple populations have reported no statistically significant increased risk of neural tube defects as a group for infants whose mothers were exposed to Zoloft during pregnancy: Louik 2007, OR 0.8 (95% CI: 0.1-6.3); Malm 2011, OR 1.77 (95% CI: 0.43-7.21); and Jimenez-Solem 2012, which reported no exposed cases. Dr. Bérard ignores these data, which refute her opinion. She also ignores that no study has reported a statistically significant increased risk for neural tube defects as a group in infants born to women exposed to Zoloft during pregnancy.

(2) Anencephaly

Dr. Bérard cites only one piece of data for an association between Zoloft and anencephaly from Alwan 2007. However, she fails to note that this finding was not confirmed in Louik 2007 (or any other study), was based only on four observed cases, and was likely a chance finding. Thus, the available evidence does not support an association between anencephaly and Zoloft.

(3) Spina Bifida

Dr. Bérard fails to cite the results from the Alwan 2007 study, which found no statistically significant association between Zoloft use and spina bifida: OR 1.2 (95% CI: 0.4-3.5). She also ignores that no study has reported a statistically significant increased risk for spina bifida in children whose mothers were exposed to Zoloft during pregnancy. Thus, the available evidence does not support an association between Zoloft and spina bifida.

i. Zoloft and Orofacial Clefts

The epidemiologic evidence does not support an association between Zoloft and orofacial clefts. Dr. Bérard cites only one piece of data for Zoloft and cleft palate, the nonsignificant findings of Malm 2011 for cleft palate alone (OR 1.42, 95% CI: 0.35-5.76). She does not cite the results from Malm for cleft lip with or without cleft palate (OR 1.01, 95% CI 0.14-7.20), nor does she cite the other non-confirmatory findings that refute her opinion: Alwan 2007: OR 0.9 (95% CI: 0.4-2.0) – cleft lip with or without cleft palate; Louik 2007: OR 1.1 (95% CI: 0.3-3.8) – cleft lip with or without cleft palate; Malm 2011: OR 1.01 (95% CI: 0.14-7.20) – cleft lip with or without cleft palate; Alwan 2007: OR 0.6 (95% CI: 0.2-1.9) – cleft palate only; Louik 2007: no cases in exposed group – cleft palate only; and Jimenez-Solem 2012: OR 0.88 (95% CI: 0.12-6.24) – orofacial cleft. Given these data and all of the other considerations noted in my report previously, the available evidence does not support an association between Zoloft and cleft lip with or without cleft palate, or cleft palate alone, or of any other orofacial clefts.

j. Zoloft and Limb Defects

Dr. Bérard discusses all limb malformations together in her report. It is problematic to consider limb malformations as a group, as these malformations (like heart defects) are particularly heterogeneous: "Epidemiologists sometimes use poor judgment when grouping malformations. As an example, limb reduction defects are frequently studied with regard to their association with environmental teratogens. In many of the studies, limb defects that are clearly related to problems of organogenesis are lumped with congenital amputations. Yet it is unlikely that any agent will be responsible for both types of malformation." (Beckman DA and Brent RL, Basic principles of developmental toxicology. In: *Medicine of the Fetus and Mother*, 2nd ed., Philadelphia, PA, Lippincott-Raven, 1999:281-88).

Nonetheless, the epidemiologic evidence demonstrates no association for any of the malformations that Dr. Bérard mentions in this section of her report. Dr. Bérard cites only one study (Louik 2007) regarding limb-reduction defects. She does not cite any of the other data for Zoloft from other studies that did not confirm Louik's findings: Alwan 2007: OR 1.2 (95% CI: 0.4-4.0) – transverse limb deficiencies; and Jimenez-Solem 2012: OR 1.00 (95% CI: 0.55-1.81) – congenital malformation of the limbs. She similarly ignores that the epidemiological evidence shows no association with musculoskeletal defects: Malm 2011: OR 0.97 (95% CI: 0.50-1.88); Colvin 2011: OR 0.69 (95% CI: 0.33-1.46); Jimenez-Solem 2012: OR 0.83 (95% CI: 0.12-5.9).

The only study she cites for club foot (Louik 2007) had, as discussed above, numerous methodological flaws including bias, and in any event found no statistically significant increased risk. No other study has reported an increased risk for club foot in infants whose mothers were exposed to Zoloft during pregnancy.

Given these data and all of the other considerations noted in my report previously, the available evidence does not support an association between Zoloft and the malformations discussed in the limb defect section of Dr. Bérard's report.

k. Zoloft and Other Defects

Dr. Bérard cites other, various congenital malformations and states that SSRIs cause these malformations. However, this approach also does not consider the errors inherent in the study designs, including confounding, ascertainment bias, and Type I error. In addition, 10 of the 12 ORs presented are not statistically significant. The two that are, anomalies of the vascular system and cystic kidney disease, come from the same study. Equally importantly, none of the results presented are for Zoloft specifically. Moreover, Dr. Bérard fails to cite multiple negative findings regarding other malformations, many of which I note above in my discussion of the studies.

Given these data and all of the other considerations noted in my report previously, the available evidence does not support an association between Zoloft and other defects.

D. Bradford Hill

Both Drs. Bérard and Vekemans mention the Bradford Hill criteria. It is important to note that Bradford Hill states in his original paper that that there should be a "clear-cut" association established and that it should be "beyond what we would care to attribute to the play of chance" prior to applying his criteria. (Bradford Hill A, The environment and disease: Association or causation?, *Proc Royal Soc Med*, 1965;58:295-300) Thus, in my opinion, the Bradford Hill criteria are not even applicable to the question of Zoloft and birth defects because there is no demonstrated association between Zoloft and birth defects. Nonetheless, I will address the components of the Bradford Hill criteria used by Drs. Bérard and Vekemans.

Strength of Association. As discussed above, an association between Zoloft and birth defects or PPHN has not been established. Therefore, this first Bradford Hill criteria would clearly not even be applicable and use of his additional criteria are inappropriate. However, even if one were to simply look at the selected odds ratios cited by Drs. Bérard and Vekemans, and ignore the play of chance or biases that are likely to have created those odds ratios, the actual ORs do not rise to the level of a strong association.[15] The strength of association criteria would not be met even if one assumes that there are any associations to begin with, which has not been established.

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[15] Shepard's Catalog of Teratogenic Agents suggests that a relative risk of 6 or more should be present as evidence of an association to be suspected. (Shepard TH, *Catalog of Teratogenic Agents*, 13th ed., Baltimore, MD, The Johns Hopkins University Press, 2010:411)

Consistency. As noted above, there is a lack of consistency across all of the associations between Zoloft and birth defects or PPHN. In addition, Bradford Hill acknowledges that one could have consistency in results due to a consistent bias in the studies used to obtain those results. Bradford Hill emphasizes the need for consistency across different study designs and approaches that can address different aspects of the association. Here, different study designs have been performed and reveal that the findings on which Drs. Bérard and Vekemans rely are due to biases. This criteria is not satisfied.

Specificity. Clearly the defects noted among women who use SSRIs are also noted among women who do not use SSRIs. It also is unlikely that a single drug could cause a non-specific range of defects that span gestational timing from preconception to late pregnancy. There is nothing specific about these disorders to SSRIs.

Temporality. Most studies attempted to identify SSRI exposure that preceded the development of birth defects. However, it is possible that some women started taking their SSRI after the critical period of organogenesis, which would violate the assumption of temporality.

Biological gradient. This refers primarily to a dose-response or duration-response relationship. As noted above, there is no evidence for either for Zoloft, and, in fact, the available evidence finds no overall dose-response (Jimenez-Solem) or duration-response (Ramos 2008).

Plausibility. Bradford Hill does not place great emphasis on this criterion. He states: "What is biologically plausible depends upon the biological knowledge of the day." This means both that lack of biological plausibility may not be useful but also that what we think is biologically plausible today may in fact be wrong. Nonetheless, it is implausible that a single drug could cause the range of birth defects purported by Drs. Bérard and Vekemans.

Coherence. Coherence refers to consistency of biological findings. That is, the data should not conflict with generally known facts of the disease of interest. This is a very difficult criteria to apply and one that is open to tremendous interpretation. Nonetheless, to my knowledge, SSRIs have not been shown to be associated biologically with birth defects in animal models in usual doses. As one reviewer noted recently: "Animal reproductive studies in rats and rabbits administered paroxetine, fluoxetine, or sertraline during organogenesis did not show a teratogenic effect." (Diav-Citrin O et al., Selective serotonin reuptake inhibitors in human pregnancy, *Obstet Gynecol Int*, 2012;2012:698947)

Experiment. There is no experimental (i.e., randomized trial) evidence available for SSRIs.

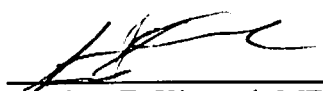
Analogy. There is, to my knowledge, no analogous data that would support SSRIs as a cause of birth defects.

My assessment is consistent with the published literature. As one article noted, "the overall current scientific evidence has not fulfilled the criteria for proof of human teratogenicity of SSRIs." (Diav-Citrin 2012).

VIII. Conclusion

For the foregoing reasons, there is no evidence of a causal association between Zoloft and any birth defects or PPHN. Plaintiffs' experts employed flawed methodologies to reach their opinions, as explained above. I reserve the right to update my opinion if new information becomes available, and I also reserve the right to use graphics and demonstratives to explain and illustrate the subjects discussed in my report.

Dated: September 12, 2013



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Schedule of Deposition Testimony in the Past Four Years

- Deposition in Woodson et al. v. Abousy et al., No. CL-2007-5870 (Circuit Court for the County of Fairfax, Virginia)
- Depositions in Fosomax cases on 2/1/13 and 5/22/2013
- No trial testimony

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100. Wogelius P; Norgaard M; Gislum M; Pedersen L; Munk E; Mortensen PB; Lipworth L; Sorensen HT | Maternal Use of Selective Serotonin Reuptake Inhibitors and Risk of Congenital Malformations | Epidemiology | 17(6):701-04 | 11/00/2006

101. Yonkers KA; Wisner KL; Stewart DE; Oberlander TF; Dell DL; Stotland N; Ramin S; Chaudron L; Lockwood C | The Management of Depression During Pregnancy: A Report from the American Psychiatric Association and the American College of Obstetricians and Gynecologists | Gen Hosp Psychiatry | 31(5):403-13 | 09/00/2009

UNIVERSITY OF PENNSYLVANIA - PERELMAN SCHOOL OF MEDICINE
Curriculum Vitae

Date: 9/12/13

Stephen E. Kimmel, MD, MSCE

Address: University of Pennsylvania School of Medicine
Center for Clinical Epidemiology and Biostatistics
Room 717, Blockley Hall
423 Guardian Drive
Philadelphia, PA 19104-6021 USA

If you are not a U.S. citizen or holder of a permanent visa, please indicate the type of visa you have:
none (U.S. citizen)

Education:

1984	A.B.	Princeton University, Princeton, NJ (Chemistry)
1988	M.D.	New York University School of Medicine, New York, NY (Medicine)
1995	M.S.C.E.	Perelman School of Medicine University of Pennsylvania (Clinical Epidemiology)

Postgraduate Training and Fellowship Appointments:

1988-1989	Intern, Internal Medicine, Brigham and Women's Hospital, Boston, MA
1989-1991	Resident, Internal Medicine, Brigham and Women's Hospital, Boston, MA
1991-1994	Fellow in Cardiology, Perelman School of Medicine University of Pennsylvania, Philadelphia, PA
1992-1994	Fellow in Pharmacoepidemiology, Perelman School of Medicine University of Pennsylvania, Philadelphia, PA

Faculty Appointments:

1994-2003	Assistant Professor of Medicine, University of Pennsylvania School of Medicine
1995-2003	Assistant Professor of Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)
2003-2011	Associate Professor of Epidemiology in Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)
2003-2011	Associate Professor of Medicine, University of Pennsylvania School of Medicine
2011-present	Professor of Epidemiology in Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)
2011-present	Professor of Medicine, University of Pennsylvania School of Medicine

Hospital and/or Administrative Appointments:

1994-2005	Attending, Hospital of the University of Pennsylvania
1994-present	Attending, Veterans Affairs Medical Center
1994-present	Director, Cardiovascular Epidemiology, Cardiovascular Division, Department of Medicine, Perelman School of Medicine University of Pennsylvania
1997-2005	Director, Epidemiology Track, Master of Science in Clinical Epidemiology Program, Perelman School of Medicine University of Pennsylvania
2002-2005	Co-Director, Master of Science in Clinical Epidemiology Program, Perelman School of Medicine University of Pennsylvania
2006-2012	Deputy Director, Clinical Epidemiology Unit, Perelman School of Medicine University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics
2011-present	Director, Center for Therapeutic Effectiveness Research, Perelman School of Medicine University of Pennsylvania
2012-present	Acting Director of the Division of Clinical Epidemiology and the Clinical Epidemiology Unit, Perelman School of Medicine University of Pennsylvania

Other Appointments:

1994-present	Senior Scholar, Perelman School of Medicine University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics (CCEB)
1997-present	Member, Graduate Group in Epidemiology and Biostatistics, Biomedical Graduate Studies Program, Perelman School of Medicine University of Pennsylvania
2003-present	Senior Fellow, Leonard David Institute
2011-present	Senior Fellow, Center for Behavioral Health Research, University of Pennsylvania

Specialty Certification:

1991	Board Certified in Internal Medicine (recertified in 2001)
1996	Board Certified in Cardiology (recertified in 2001)
2001	American College of Epidemiology

Licensure:

1991-Present	Commonwealth of Pennsylvania

Awards, Honors and Membership in Honorary Societies:

1984	Phi Beta Kappa
1984	Sigma Xi Research Society
1984	Sigma Xi Award for Outstanding Research in Chemistry
1988	Alpha Omega Alpha

1993	American College of Cardiology/Merck Adult Cardiology Fellowship Award
1996	Award for Best Poster, International Conference on Pharmacoepidemiology
2001	Excellence in Teaching in Epidemiology Award, Master of Science in Clinical Epidemiology Program
2001	Leonard Berwick Memorial Teaching Award
2003	ISPE Pharmacoepidemiology and Drug Safety Best Paper Prize
2004	UPHS Quality and Safety Award for Controlling Hypertension at Penn (CHAP)
2007-2008	Excellence in Teaching Epidemiology Award
2009	Honorable Mention, ISPE Pharmacoepidemiology and Drug Safety Best Article Award - "Moderate and high affinity serotonin reuptake inhibitors increase the risk of upper gastrointestinal toxicity"
2010-2011	Lindback Award for Distinguished Teaching

Memberships in Professional and Scientific Societies and Other Professional Activities:

International:

1994-Present	International Society of Pharmacoepidemiology (Member 1994-2004, Fellow 2004-present)
1998-2001	ISPE Educational Committee (Member)
2000-2001	ISPE Board of Directors (Member)

National:

1984-Present	Sigma Xi Research Society (Member)
1996	American College of Cardiology (Member, Subcommittee to Write Statement on "Development & Maintenance of Competence in Coronary Intervention Procedures")
1996-Present	American College of Cardiology (Fellow)
1996-Present	American Heart Association Council on Epidemiology and Prevention (Member)
1996-Present	American Heart Association and American Heart Association Council on Epidemiology and Prevention (Fellow)
1996-Present	Society for Cardiac Angiography and Interventions (Elected Consultant)
1998-1999	American College of Cardiology Scientific Sessions (Chair of Session)
1999-Present	Society for Epidemiologic Research (SER) (Member)

2000-Present	American Society for Clinical Pharmacology and Therapeutics (ASCPT) (Member)
2000-2003	Program Committee, National American Heart Association Council on Epidemiology (Member)
2001	Advisory Group, "One of a Kind", American Heart Association Quality of Care and Outcomes Research Expert Panel (Member)
2001-Present	American College of Epidemiology (Member)
2001-2002	National Peer Review Committee, American Heart Association Outcomes Research (Member)
2001-2002	Quality of Care and Outcomes Research Network of Experts, American Health Association (Appointed Member)
2002	Advocacy Committee, Epidemiology and Prevention Council of the American Heart Association (Member)
2002-2004	American College of Cardiology, National Cardiovascular Data Registry, Scientific and Clinical Support Task Force (Member)
2002-Present	National Institutes of Health, National Heart, Lung, and Blood Study Section (Ad Hoc Member reviewing all K-grants)
2002	Session at the Genomics Revolution: Bench to Bedside to Community and the 42nd Annual Conference on Cardiovascular Disease and Epidemiology and Prevention (Chair)
2006-Present	American College of Epidemiology (Fellow)
2007-Present	American Society for Clinical Investigation (ASCI) (Member)
2008-Present	American Society of Human Genetics (Member)
2008-Present	Pharmacogenetics Research Network (PGRN) (Affiliate Membership)

Local:

2000-2002	Health Measurement Task Force, Pennsylvania Delaware, American Heart Association (Member)

Editorial Positions:

1994-Present	Editorial Consultant, Journal of General Internal Medicine
1995-Present	Editorial Consultant, Annals of Internal Medicine

1996-Present	Editorial Consultant, The Journal of the American Medical Association
1997-Present	Editorial Consultant, Journal of the American College of Cardiology
1997-Present	Editorial Consultant, American Heart Journal
1997-Present	Editorial Consultant, New England Journal of Medicine
1999-Present	Associate Editor for North America, Pharmacoepidemiology and Drug Safety
2002-Present	Editorial Consultant, American Journal of Cardiovascular Drugs
2002-Present	Editorial Consultant, American Journal of Cardiology
2002-Present	Editorial Consultant, Circulation
2002-Present	Editorial Consultant Archives of General Psychiatry
2008-Present	Editorial Consultant, Blood

Academic and Institutional Committees:

1997-2005	Member, Master of Science in Clinical Epidemiology Admission Committee
1997-Present	Member, CCEB Graduate Teaching Curriculum Committee
1998-2002	Member, BGS Curriculum/Academic Standards Committee
1998-2005	Member, PhD in Epidemiology Admission Committee
1998-2002	Chair, CCEB Awards Committee
1999-2001	Chair, CCEB Institutional Review Board Committee
2002-2005	Chair, Master of Science in Clinical Epidemiology Curriculum Committee
2003-Present	Member, Committee on Appointments and Promotions (COAP), Department of Biostatistics and Epidemiology, University of Pennsylvania School of Medicine
2007-Present	Chair, Division of Epidemiology Recruitment Committee
2007-2008	Chair, University of Pennsylvania Bioethics External Review Committee
2008-Present	Chair of the Think Health Scientific Advisory Committee

Major Academic and Clinical Teaching Responsibilities:

1994-1996	Medicine 100 for Medical Students, Fall Semester (Penn Medicine)
1994-1998	Medicine 101A for Medical Students, Spring Semester (Penn Medicine)
1994-2000	Preceptor for EP 154, Medical Student Epidemiology Course, Spring/Fall Semester (Penn Medicine)
1994-Present	Attending Rounds at HUP and VA Medical Center (2 months/year)(Penn Medicine)
1994-Present	Clinic Supervisor, VA Medical Center and HUP (Penn Medicine)
1994-Present	Methods in Clinical Research. Seminar Series for Cardiology Fellows, Medical Residents, and Medical Students (Penn Medicine)
1994-Present	Faculty Preceptor, Cardiology Journal Club (Penn Medicine)
1998-Present	EP644 Cardiopulmonary Epidemiology, Advanced Master of Science in Clinical Epidemiology Course: Course Director and Teacher, Summer Semester (Penn Medicine)

2002-2005	Co-Director, MSCE Program at the University of Pennsylvania School of Medicine (30-40 Masters Students/year)
2006	"Adherence from the Academic Perspective. Setting a Research and Action Agenda to Increase Healthy Behaviors and Adherence.", Leonard Davis Institute, University of Pennsylvania, Philadelphia, PA
2008	"Warfarin Pharmacogenetics", The Children's Hospital of Philadelphia Pediatric Care Research Lecture, Philadelphia, PA

Lectures by Invitation:

Oct, 1995	"Calcium Channel Blockers: What Should We Do in the Meantime?"-Medicine Grand Rounds, Montgomery Hospital, Norristown, PA
May, 1996	"Current Controversy with Calcium Channel Blocker Use: What do we do now? Lehigh Valley Hospital Regional Symposium Series, Sixteenth Annual Update in Cardiology, Lehigh Valley Hospital, Allentown, PA
Jul, 1996	"Calcium Channel Blockers: What Should We Do in the Meantime?" - Medical Grand Rounds, Hospital of the University of Pennsylvania, Philadelphia, PA
Oct, 1996	"Calcium Channel Blockers: What Should We Do in the Meantime?" - Medical Grand Rounds, Chestnut Hill Hospital, Philadelphia, PA
Dec, 1996	"Current Controversy Over Calcium Channel Blockers" - Mid-Atlantic Chapter of the American College of Clinical Pharmacy, Philadelphia, PA
Jan, 1997	"Aspirin and 'Primary' Prevention of Cardiovascular Disease" - US Food and Drug Administration, Gaithersburg, MD
May, 1998	"Drug Safety Case Reports: From Calcium Blockers to FenPhen" - American Society of Hypertension, 13th Annual Meeting, New York, NY
Jun, 1999	"Sexual Activity and Cardiac Risk - Epidemiology" - International Consensus Conference - Sexual Activity and Cardiac Risk, Princeton, NJ
Dec, 1999	"The Health Risks of Obesity" - 1999 AHPA Ephedra International Symposium, Arlington, VA
Dec, 1999	"Coronary Stents", Cardiology Grand Rounds, Hospital of the University of Pennsylvania", Philadelphia, PA
Mar, 2000	"Clinical Epidemiology-What Qualifies As A Valid Study and What Isn't Ready For Prime Time?" ACC Medical Writers Symposium (in conjunction with the American College of Cardiology 49th Annual Scientific Session), Anaheim, CA
Aug, 2000	"Review of Available Data on Ephedra Alkaloids" Department of Health and Human Services Office of Women's Health, Washington, DC

Sep, 2000	"Coronary Stents: The Good, the Bad, or the Ugly?" Northwestern University School, Cardiology Grand Rounds, Chicago, IL
Mar, 2001	"Common Sense and Statistics in Identifying the High-Risk Patient" American College of Cardiology 50th Annual Scientific Session (ACC 2001), Orlando, FL
Mar, 2001	"Clinical Outcomes Research in Cardiology: A Broad Range of Research Opportunities" 41st Annual Conference on Cardiovascular Disease Epidemiology and Prevention, San Antonio, TX
Nov, 2001	"Non-Steroidal Anti-Inflammatory Medications and Myocardial Infarction: Study Designs Issues" Epidemiology Advisory Panel, Pfizer, New York, NY
Nov, 2001	"Volume-Outcome Relationship for PCI and CABG: Lessons from Registries" 2001 American Heart Association Scientific Sessions, Anaheim, CA
Jan, 2002	"The Epidemiology of Antidepressant Therapy" Duke University Depression and Cardiovascular Disease Meeting, Baltimore, MD
Mar, 2002	"Assisting Trained Clinicians to Become Researchers" 60th Association of Teachers of Preventive Medicine Annual Meeting, Washington, DC
Jun, 2002	"Volume and Outcomes in Primary Angioplasty for Acute MI." Ohio-American College of Cardiology Annual Meeting, Huron, OH.
Jun, 2002	"NSAIDs and COX-2 Inhibitors: Good for You, or Dangerous?" Hospital of the University of Pennsylvania, Cardiology Grand Rounds, Philadelphia, PA.
Oct, 2002	"NSAIDs, Aspirin and COX-2 Inhibitors: Risky Business or Unexpected Benefits?" University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics Seminar, Philadelphia, PA
Nov, 2002	"Selection of Pharmacological Approaches for Patients with Cardiovascular Disease and Depression." 2002 American Heart Association Scientific Sessions, Chicago, IL.
Apr, 2003	"NSAIDs, COX-2 Inhibitors, and Cardiovascular Disease: Balancing the Potential Benefits and Risks." Stanford University, Stanford, CA
Nov, 2003	"SSRIs and Cardiovascular Disease." University of Pennsylvania Symposium. Psychiatry in Medicine: Medicine in Psychiatry, Philadelphia, PA
Sep, 2004	"Improving Anticoagulation Care." The Agency for Healthcare Research and Quality (AHRQ) Third Annual Patient Safety Research Conference, Arlington, VA
Nov, 2004	"Molecular Pharmacoepidemiology", GlaxoSmithKline Seminar, Collegeville, PA
Jun, 2005	"Genetic and Molecular Pharmacoepidemiology", DIA 41st Annual Meeting, Washington, DC

Feb, 2006	"Human Pharmacogenomic Epidemiology", National Institutes of Health, Interdisciplinary Research Centers Workshop, Bethesda, MD
Sep, 2006	"Coxibs NSAIDs and the Heart: Did We Get It Wrong?" Controversies and Conundrums in Cardiovascular Medicine 2006, 23rd Annual Santa Fe Colloquium on Cardiovascular Therapy, Santa Fe, NM
Dec, 2007	"Warfarin Pharmacogenomics: Ready for prime Time?" University of Connecticut Cardiology Grand Rounds, Farmington, CT
Mar, 2008	"Warfarin Pharmacogenomics: The FDA and the Science" Cardiology Grand Rounds, University of Colorado, Denver, CO
May, 2008	"Measured vs. Assumed Drug Dosing Histories in the Management of Oral Anticoagulation: Doses & INR Levels" Drug Information Association (DIA), Washington, DC
May, 2008	"Methods and Approaches to Clinical Research in Adult Congenital Heart Disease" Research Symposium, 2008 Fifth National ACHA Conference, Philadelphia, PA
Sep, 2008	"Key research and policy issues to improve outcomes of anticoagulation therapies" AHRQ Centers for Education and Research on Therapeutics 7th Annual Partnerships to Advance Therapeutics Meeting, Bethesda, MD
Oct, 2008	"Warfarin Pharmacogenetics: Challenges and Opportunities" Pharmacogenetics Research Network (PGRN) Scientific and Steering Committee Meeting, University of North Carolina, Chapel Hill, NC
Nov, 2008	"Perspective on Genotype Guiding of Warfarin", Critical Path Institute's Warfarin Summit II, Bethesda, MD
Aug, 2009	"Warfarin", Introduction to Pharmacogenetic Epidemiology, 25th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Rhode Island Convention Center, Providence, RI
Aug, 2009	"Warfarin as a Prototype Pharmacogenetic Example", Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 25th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Rhode Island Convention Center, Providence, RI
Oct, 2009	"Design of the Clarification of Optimal Anticoagulation through Genetics (COAG) Trial", Inaugural Meeting of the Genomic Applications in Practice and Prevention Network (GAPPNet), sponsored by the CDC Office of Public Health Genomics, Ann Arbor, MI

Aug, 2010	"Warfarin as a Prototype Pharmacogenetic Example," Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 26th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Brighton, UK
Aug, 2010	"Warfarin Pharmacogenetics: A Clinical Trial Perspective", Duke Clinical Research Institute, Pharmacogenomics in Cardiovascular Disease: Balancing Scientific Promise with Clinical Reality, McLean, VA
Oct, 2010	"Adherence and Novel Methods", Duke Clinical Research Institute, Medication Adherence: What do we need to do to get people to take their medicines? McLean, VA
Oct, 2010	"Warfarin Pharmacoepidemiology", The Eighth International Workshop on Pharmacodynamics of Anticancer Agents, Hakone, Japan
Nov, 2010	"How Do We Bring Pharmacogenetics into Practice?", FIP Pharmaceutical Sciences 2010 World Congress, AAPS Annual Meeting and Exposition, New Orleans, LA
Nov, 2010	"Effectiveness and Safety Research: Translation Into Practice", Kaiser Permanente, San Francisco, CA
Apr, 2011	"New incentive approaches for adherence." University of Pennsylvania, Emerging Statistical Issues in the Conduct and Monitoring of Clinical Trials, University of Pennsylvania Annual Conference on Statistical Issues in Clinical Trials, Philadelphia, PA
Aug, 2011	"Warfarin as a Prototype Pharmacogenetic Example," Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 27th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Chicago, IL
Apr, 2012	"Personalized Medicine: The Promise and the State of the Science" University of the Sciences, Making the Connections: Personalized Medicine - From Promise to Public Health and Policy Symposium and panel discussion, Philadelphia, PA
Apr, 2012	"Therapeutic Effectiveness Research or Why "We Can't Solve Problems By Using the Same Kind of Thinking We Used When We Created Them."" Hospital of the University of Pennsylvania Cardiology Grand Rounds, Philadelphia, PA
Aug, 2012	"Pharmacogenetics: Clinical Utility Studies and Other True-Life Examples" Introduction to Pharmacogenetic Epidemiology, 28th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Barcelona, Spain
Apr, 2013	"Why We Are Failing to Deliver Optimal Medical Care: A Case for Therapeutic Effectiveness Research" Northwestern University Grand Rounds, Chicago, IL
Apr, 2013	"Personalized Medicine in Cardiology", Utrecht University Symposium - The future of Anticoagulation Therapy, Amsterdam, Netherlands

May, 2013	"Bringing Pharmacogenetics into Practice", Children's Hospital of Philadelphia Pediatric Cardiology Research Lecture, Philadelphia, PA

Organizing Roles in Scientific Meetings:

2001	Member, Program Committee, National American Heart Association Council on Epidemiology New Orleans, LA
2002	Member, Program Committee, National American Heart Association Council on Epidemiology Honolulu, HI
2003	Member, Program Committee, National American Heart Association Council on Epidemiology Miami, FL
2006	ISPE, International Conference on Pharmacoepidemiology, Committee on Pharmacogenomics Lisbon, Portugal
2009	ISPE Special Interest Group in Pharmacogenomics Providence, RI
Nov, 2010	Session Chair, "Pharmacogenetics: Found in Translation?", FIP Pharmaceutical Sciences 2010 World Congress, AAPS Annual Meeting and Exposition New Orleans, LA
2010	"Medication Adherence: Specific Recommendations to Transform Clinical Care" Session II Co-Chair, Duke Clinical Research Institute, Medication Adherence: What do we need to do to get people to take their medicines? McLean, VA

Grants:

Pending:

Funds for Genomic Medicine Meetings , NIH/NHGRI, U01, 1/2014-12/2016 (Kimmel S, PI), $267,768/annual direct costs, 2.5% effort (Role in grant: PI, This supplement will provide funds for 4yrs, to facilitation and cost of 3 Genomic Medicines meetings per year for 4 years, to be organized in conjuction with NHGRI staff.)

A Randomized Trial of Copayment Reductions and Lottery Based Financial Incentives, NIH, R01-HS022657, 10/2013-9/2018 (Doshi J, PI), $498,642/annual direct costs, 2.5% effort (Role in grant: Co-Investigator, The proposed study will evaluate the effectiveness and cost-effectiveness of two innovative approaches for improving cardioprotective medication adherence. We will assess the effectiveness of a daily lottery-based financial incentive on cardioprotective medication adherence relative to the control group over 9 months.)

Current:

Genomic Medicine Pilot Demonstration Projects Coordinating Center, NIH/NIHGRI, U01-HG007266 , 6/2013-4/2017 (Kimmel SE, PI), $400,000/annual direct costs, 23% effort (Role in grant: PI, The goal of the Coordinating Center at the University of Pennsylvania is to ensure the success of individual Genomic Demonstration Pilot Projects while stimulating collaboration across projects to produce and disseminate generalizable knowledge that can be used to expand and sustain existing efforts and to promote implementation in other settings and for other diseases.)

Patient and Provider Perspectives on Reasons for Hospital Readmissions, Patient Centered Outcomes Research Institute (PCORI), IP2-P10000186, 10/2012-12/2014 (Kimmel, S., PI), $249,925/annual direct costs, 15% effort (Role in grant: PI, Patients with chronic illnesses that require hospitalization, such as heart failure, often have difficulty managing their diseases after discharge and are often readmitted to the hospital within a short period. Efforts to predict and reduce readmissions have not been tailored to the specific needs of patients most at risks for readmission.)

Clinical Importance of Drug-Drug-Interactions, NIH, R01-AG025152, 4/2012-3/2017 (Hennessy, S., PI: Kimmel, S., Co-Investigator), $479,890/annual direct costs, 2% effort (Role in grant: Co-Investigator, Propose to extend our approach to population DDI studies in 2 ways: 1) by incorporating mechanistic considerations, increasing translation to/from the "left hand" side of the translational spectrum; 2) implementing a sequential approach of identifying/quantifying risk, then, based on this step, characterizing important DDIs, with the goal of providing clinical detail needed for future efforts to mitigate risk;)

Clinical Outcomes of Saxagliptin, AstraZeneca and Bristol-Myers Squibb, 8/2010-8/2017 (Strom, B., PI: Kimmel, S., Co-Investigator), $1,032,225/annual direct costs, 1% effort (Role in grant: Co-Investigator, This project will provide information on utilization and outcomes of saxagliptin.)

The Risk of Myocardial Infarction in Patients with Psoriasis, NIH, R01-HL099744, 3/2009-1/2014 (Gelfand, J., PI), $499,195/annual direct costs, 10% effort (Role in grant: Co-Investigator, The goal of this project is to determine the risk of MI and stroke in patients with psoriasis.)

Genetic and Environmental Determinants of Warfarin Response, Sub to UAB/NIH, R01 HL092173, 5/2008-4/2014 (Limdi, N., PI: Kimmel, S., Co-Investigator), $44,468/annual direct costs, 9% effort (Role in grant: Co-PI, The primary objective of the study is to quantify the association between warfarin dose requirements and genetic variants to develop and validate dosing algorithms.)

Randomized Trial of Genotype-Guided Dosing of Warfarin Therapy, NIH, N01, 4/2008-1/2014 (Kimmel, S., PI), $1,674,325/annual direct costs, 30% effort (Role in grant: PI, The primary aim of this program project grant is to identify predictors of adherence with warfarin therapy and develop a prediction rule to identify patients at risk for poor adherence with warfarin.)

Continuation of the Chronic Renal Insufficiency Cohort (CRIC) Study, NIH-NIDDK, U01-DK060990, 9/2001-4/2014 (Feldman, H., PI), $1,988,801/annual direct costs, 2% effort (Role in grant: Co-Investigator, The objective of this study is to focus on the prediction and mechanisms of progressive renal disease and cardiovascular events in patients with CRI.)

Prediction of Warfarin Dosing Using Clinical and Genetic Factors., NIH, R01-HL066176, 3/2001-6/2014 (Kimmel, S., PI), $515,039/annual direct costs, 2% effort (Role in grant: PI, A prospective cohort study that will incorporate patient, environmental factors, and genetic variants to develop and validate dose prediction models.)

Past:

Development of Novel Methods for Comparative Effectiveness Clinical Trials, NCRR, UL1-RR024134 , 8/2011-6/2013 (Kimmel, S., PI), $183,060/annual direct costs, 15% effort (Role in grant: PI, Comparative Effectiveness research (CER) of therapeutic strategies must often serve a dual role: Be rigorous yet also relevant to clinical practice. Often, a decision is made to sacrifice one for the other. Although clinical trials are ideal for producing unbiased comparative effectiveness results, they typically study a single intervention at a single point in time and thus do not inform the way that many existing therapies are used in actual clinical practice.)

Adherence to Antidepressant Medication and Hypertension Treatment, NIH, R34, 12/2009-7/2013 (Bogner, H., PI), $148,936/annual direct costs, 1% effort (Role in grant: Co-Investigator, The primary aims of this proposal are to design & test an integrated intervention strategy.)

Data Infrastructure for Post-marketing Comparative Effective, NIH, RCI-AG025152, 9/2009-8/2012 (Hennessy, S., PI), $332,891/annual direct costs, 1% effort (Role in grant: Co-Investigator, To demonstrate the utility of this data resource for comparative effectiveness research by examining the comparative effectiveness of different strategies for initial supplementation and monitoring of potassium (K) in patients newly starting loop or thiazide diuretics with regard to preventing a) all-cause death and b) sudden death/ventricular arrhythmia.)

Developing Interactive Technologies to Improve Research and Health Behavior , NIH, RC2-AG036592, 9/2009-9/2011 (Asch, D., PI: Volpp, K., Co-Investigator), $1,757,058/annual direct costs, 2% effort (Role in grant: Co-Investigator)

A Randomized Trial of Interventions to Improve Warfarin Adherence, NIH, R01-HL090929, 9/2008-5/2012 (Kimmel, S., PI), $499,312/annual direct costs, 1.5% effort (Role in grant: PI, A randomized trial to evaluate the effectiveness and cost-effectiveness of two novel approaches aim to improve anticoagulation control by increasing warfarin adherence.)

Cardiovascular Safety of Stimulant Medications for Attention Deficit Hyperactivity Disorder, Shire, N/A, 3/2008-6/2011 (Hennessy, S., PI), $540,789/annual direct costs, 2.5% effort (Role in grant: Co-Investigator)

Lottery Based Approach to Improving Warfarin Adherence, Penn/CMA Aetna, 9/2007-8/2009 (Kimmel, S., PI), $398,994/annual direct costs, 1% effort (Role in grant: Co-PI)

Clinical Risk Factors for Primary Graft Dysfunction, NIH, R01-HL087115, 8/2007-7/2012 (Christie, J., PI), $632,632/annual direct costs, 2.5% effort (Role in grant: Collaborator, We aim to study the association of candidate genes in donors and recipients with subsequent development of PGD.)

Collaboration to Reduce Disparities in Hypertension, Pfizer, 3/2007-2/2009 (Kimmel, S., PI), $667,677/annual direct costs, 1% effort (Role in grant: PI)

Genetics of Primary Graft Dysfunction, NIH, R01-HL081619, 2/2007-1/2012 (Christie, J., PI), $574,259/annual direct costs, 2.5% effort (Role in grant: Co-Investigator, We aim to study the association of candidate genes in donors and recipients with subsequent development of PGD.)

Incidence and Predictors of Abnormal Liver Associated Enzymes in Patients with Atrial Fibrillation in a Routine Clinical Care Population, GlaxoSmithKline, 9/2005-5/2006 (Lewis, J., PI), $151,642/annual direct costs, 10% effort (Role in grant: Co-PI)

Drug-Induced Sudden Death & Ventricular Arrthythmia, NIH, R01 HL076697, 1/2005-11/2009 (Hennessy, S., PI), $458,173/annual direct costs, 2.5% effort (Role in grant: Co-Investigator)

Transdisciplinary Research on Genetics of Complex Traits, NIH, 9/2004-7/2009 (Kimmel, S., PI), $380,039/annual direct costs, 1% effort (Role in grant: PI)

Evaluation of the Controlling Hypertension at Penn (CHAP) Pilot Project, NIH, 1/2004-12/2005 (Sean Hennessy, PharmD, PI), $138,028/annual direct costs, 2% effort (Role in grant: Co-Investigator)

Value of Electronic Clinical Records for Medical Research, NIH, 7/2003-6/2008 (Tannen, R., PI), $415,159/annual direct costs, 5% effort (Role in grant: Co-Investigator)

Collaboration to Reduce Disparities in Hypertension, PA Department of Health, 3/2003-2/2007 (Kimmel, S., PI: Volpp, K., Co-Investigator), $1,001,253/annual direct costs, 1% effort (Role in grant: PI)

A Prediction Rule for Post-Coronary Artery Bypass Grafting (CABG) Supraventricular Tachycardias, NIH, 12/2002-6/2007 (Rod Passman, MD, PI), $74,625/annual direct costs (Role in grant: Collaborator)

COX-2 Inhibitors and Cardiac Outcomes, Merk & Co, Inc., 11/2002-4/2003 (Stephen E. Kimmel, MD, MSCE, PI), $250,000/annual direct costs, 2% effort (Role in grant: PI)

Patient Oriented Research in Anticoagulation, NIH, 9/2002-8/2008 (Kimmel, S., PI), $123,894/annual direct costs, 50% effort (Role in grant: PI)

Selective Serotonin Reuptake Inhibitors and Cardiac Outcomes, Glaxo-SmithKline Beecham, 1/2002-1/2003 (PI, PI), $163,881/annual direct costs, 2% effort (Role in grant: PI)

Prospective Cohort Study of Chronic Renal Insufficiency, NIH, 9/2001-6/2008 (Feldman, H., PI), $790,753/annual direct costs, 3.5% effort (Role in grant: Co-Investigator)

Improving Patient Safety by Reducing Medication Errors, NIH, 9/2001-8/2006 (Strom, B., PI), $867,334/annual direct costs, 15% effort (Role in grant: Project Leader)

Heart Sense: A Game for Reducing Heart Attack Pre-Hospitalization Delay-Phase II, NIH, 9/2001-8/2003 (Silverman, PI), $54,115/annual direct costs, 8% effort (Role in grant: Co-Investigator)

Neurohormonal Activation in Pulmonary Hypertension, NIH, 7/2001-6/2006 (Kawut, S., PI), $130,180/annual direct costs (Role in grant: Co-Investigator)

Predictors of Anticoagulation Control on Warfarin, NIH, 3/2001-1/2008 (Kimmel, S., PI), $502,197/annual direct costs, 30% effort (Role in grant: PI)

Clinical risk factors and biomarkers of oxidant stress in primary graft failure following lung transplantation, NIH, 7/2000-6/2005 (Jason Christie, MD, PI), $120,847/annual direct costs (Role in grant: Collaborator,)

Risk of Upper Gastrointestinal Bleeding with OTC NSAIDs, Bayer Consumer Care, 7/2000-12/2003 (Jim Lewis, MD, PI), $397,922/annual direct costs, 10% effort (Role in grant: Co-Investigator)

AIDS Clinical Trials Unit, NIH, 1/2000-12/2004 (Friedman, PI), $7,684,407/annual direct costs, 4% effort (Role in grant: Co-Investigator)

COX-2's and Myocardial Infarction, Searle Pharmaceutical, 1/2000-4/2003 (Stephen E. Kimmel, MD, MSCE, PI), $268,547/annual direct costs (Role in grant: PI)

Health Effects of Schizophrenia and Antipsychotic Medications, Pfizer, 12/1999-3/2001 (Sean Hennessy, PharmD, PI), $218,375/annual direct costs, 7.5% effort (Role in grant: Co-Investigator)

Protamine Mortality Study, Pharming, 11/1999-4/2001 (Stephen E. Kimmel, MD, MSCE, PI), $38,866/annual direct costs, 6% effort (Role in grant: PI)

Heart Sense: A Game for Heart Attack Symptom Training, NIH, 10/1999-9/2000 (Silverman, PI), $68,244/annual direct costs, 4% effort (Role in grant: Co-Investigator)

Early Determination of Stroke Subtype with CT Angiography, NIH, 7/1999-6/2004 (Scott Kasner, MD, PI), $132,891/annual direct costs (Role in grant: Collaborator)

Left Ventricular Geometry and Cardiac Risk in Blacks, NIH CAP Award, 7/1998-6/2003 (Martin Keane, MD, PI), $81,345/annual direct costs (Role in grant: Co-Investigator)

VA Stars and Stripes Healthcare Network Competitive Pilot Project, Veteran's Administration, 7/1998-6/2000 (Michael Berkwits, PI), $24,550/annual direct costs (Role in grant: Supervisor of PI)

Risk Factors for Valve Abnormalities Among Users of Fenfluramine or Dexfenfluramine: A Case-Series and Case Control Study, Wyeth-Ayerst, 2/1998-6/2003 (Stephen E. Kimmel, MD, MSCE, PI), $1,516,393/annual direct costs, 20% effort (Role in grant: PI,)

Prevention of Myocardial Infarction by Non-Steroidals, NIH, 9/1997-8/2002 (Stephen E. Kimmel, MD, MSCE, PI), $432,914/annual direct costs, 8% effort (Role in grant: PI)

Rates and Predictors of Repeat PTCA, COR Therapeutics, Inc., Key Pharmaceuticals, Inc., 5/1997-4/1999 (Stephen E. Kimmel, MD, MSCE, PI), $222,762/annual direct costs, 4% effort (Role in grant: PI)

Acute Myocardial Infarction and use of Nicotine Patches, Marion Merrell Dow, CIBA Pharmaceuticals & McNeil Labs, 12/1994-6/2000 (Stephen E. Kimmel, MD, MSCE, PI), $453,765/annual direct costs, 10% effort (Role in grant: PI)

Determining Incidence and Risk of Protamine Reactions, NIH, , 9/1994-8/1999 (Stephen E. Kimmel, MD, MSCE, PI), $82,000/annual direct costs, 80% effort (Role in grant: PI)

Protamine Reactions Following Cardiopulmonary Bypass, ACC/Merck Adult Cardiology Fellowship Award, 7/1993-6/1994 (Stephen E. Kimmel, MD, MSCE, PI), $35,000/annual direct costs, 80% effort (Role in grant: PI,)

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Books:

1. Kimmel SE, Rebbeck T, Leufkens HGM: Molecular Pharmacoepidemiology. Pharmacoepidemiology, Fourth Edition. Strom BL (eds.). West Sussex, England: John Wiley and Sons Ltd, Page: 571-586, 2005.

2. Strom BL, Kimmel SE: Textbook of Pharmacoepidemiology. Textbook of Pharmacoepidemiology. Strom BL, Kimmel SE (eds.). West Sussex: John Wiley. 2006.

3. Strom BL, Kimmel SE, Hennessy S: Pharmacoepidemiology. Fifth Edition. Pharmacoepidemiology. Fifth Edition. Strom BL, Kimmel SE, Hennessy S (eds.). John Wiley&Sons, Ltd, 2012.

4. Strom BL, Kimmel BL, Hennessy S: Textbook of Pharmacoepidemiology. Second Edition. Textbook of Pharmacoepidemiology. Second Edition. Strom BL, Kimmel BL, Hennessy S (eds.). West Sussex: John Wile, 2013.

Exhibit B

SOUTH EASTERN SYDNEY ILLAWARRA
NSW HEALTH

EASTERN SUBURBS
MENTAL HEALTH SERVICE
The Kiloh Centre
The Prince of Wales Hospital
Barker Street, Randwick 2031
PHN. 9382.4352 FAX. 9382.4399

Dr Matthew Large
Senior Psychiatrist and Medical Superintendent

23 October 2014

DECLARATION OF MATTHEW LARGE, B.Sc., M.B.,B.S. FRANZCP

Matthew Large, B.Sc., M.B.,B.S. FRANZCP, declares:

1. I am the senior and corresponding author of a 2013 meta-analysis of SSRI antidepressants and congenital malformations: Nicholas Myles, Hannah Newall, Harvey Ward, and Matthew Large, Systematic meta-analysis of individual selective serotonin reuptake inhibitor medications and congenital malformations, Aust N Z J Psychiatry 2013;47:1002-1012. A copy of my CV is attached.

2. I have reviewed comments of Nicholas Jewell, Ph.D., asserting that (a) the meta-analysis may not have followed its own pre-defined inclusion/exclusion criteria and should have excluded Alwan (2007) and Malm (2011), and (b) the heterogeneity observed for the sertraline (Zoloft) studies should have been investigated and discussed. Neither of Dr. Jewell's assertions is well-founded.

Inclusion/Exclusion Criteria

3. In pertinent part, the exclusion criteria in our meta-analysis specified that studies were excluded if they reported "a control group exposed to any antidepressant medication." In other words, studies were excluded if all of the women in the control group took some other antidepressant. Neither Alwan (2007) nor Malm (2011) had a control group in which all of the women were exposed to any antidepressant medication. Accordingly, Alwan (2007) and Malm (2011) were properly included in our meta-analysis in accordance with the pre-defined inclusion/exclusion criteria.

4. In addition, the exposure to other antidepressant medication in the control groups in Alwan (2007) and Malm (2011) was entirely trivial. Further to this point, we used odds ratios that were adjusted for other medications. In Malm (2011), exposure to other antidepressants is not explicitly stated, rather it says "crude and adjusted logistic regression were performed ... with independent variables ... (including) other psychiatric drugs (including antiepileptics)." As shown in Table 1 of Malm (2011), "any other psychiatric medication purchases" accounted for 21.6% of the SSRI group and 1% of the non-SSRI group. In Alwan (2007), other medications were

adjusted for in the odds by logistic regression and, as shown in Table 1, only accounted for 0.8% of the case group and 0.5% of the control group. Such negligible exposure to other antidepressants would not have affected the results.

Heterogeneity

5. While the sertraline studies that were included in our meta-analysis were more heterogeneous than studies of other SSRIs, such differences were properly addressed by the a priori use of a random-effects model. As we explained in our published study, "A random-effects meta-analysis of independent groups was used on an a priori basis for all analyses because of between-study differences in the populations and study methodology. Random-effects meta-analysis requires only that thresholds are consistently applied within studies and allows for differences in the methods and studied populations, including differences such as the thresholds for inclusion in the exposure group and differences in the definitions of major, minor, and cardiac malformations."

6. Although Dr. Jewell asserts that the meta-analysis should have investigated and discussed the source of between-study variation, with only five sertraline studies, it is not possible to do so.

7. In sum, the meta-analysis methodology was properly applied in our study and the conclusions set forth in the study are sound.

8. I have not received any compensation from Pfizer for my time in preparing this declaration.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Matthew Large, B.Sc., M.B.,B.S. FRANZCP

Dated: Randwick, Sydney, NSW 2031, Australia
October 23, 2014

Exhibit C

UNIVERSITY OF PENNSYLVANIA - PERELMAN SCHOOL OF MEDICINE
Curriculum Vitae

Date: 03/20/2015

Stephen E. Kimmel, MD, MSCE

Address: University of Pennsylvania School of Medicine
Center for Clinical Epidemiology and Biostatistics
Room 923, Blockley Hall
423 Guardian Drive
Philadelphia, PA 19104-6021 USA

If you are not a U.S. citizen or holder of a permanent visa, please indicate the type of visa you have:
none (U.S. citizen)

Education:

1984	A.B.	Princeton University, Princeton, NJ (Chemistry)
1988	M.D.	New York University School of Medicine, New York, NY (Medicine)
1995	M.S.C.E.	Perelman School of Medicine University of Pennsylvania (Clinical Epidemiology)

Postgraduate Training and Fellowship Appointments:

1988-1989	Intern, Internal Medicine, Brigham and Women's Hospital, Boston, MA
1989-1991	Resident, Internal Medicine, Brigham and Women's Hospital, Boston, MA
1991-1994	Fellow in Cardiology, Perelman School of Medicine University of Pennsylvania, Philadelphia, PA
1992-1994	Fellow in Pharmacoepidemiology, Perelman School of Medicine University of Pennsylvania, Philadelphia, PA

Faculty Appointments:

1994-2003	Assistant Professor of Medicine, University of Pennsylvania School of Medicine
1995-2003	Assistant Professor of Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)
2003-2011	Associate Professor of Epidemiology in Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)
2003-2011	Associate Professor of Medicine, University of Pennsylvania School of Medicine
2011-present	Professor of Epidemiology in Biostatistics and Epidemiology, University of Pennsylvania School of Medicine (Secondary)

2011-present	Professor of Medicine, University of Pennsylvania School of Medicine

Hospital and/or Administrative Appointments:

1994-2005	Attending, Hospital of the University of Pennsylvania
1994-present	Attending, Veterans Affairs Medical Center
1994-present	Director, Cardiovascular Epidemiology, Cardiovascular Division, Department of Medicine, Perelman School of Medicine University of Pennsylvania
1997-2005	Director, Epidemiology Track, Master of Science in Clinical Epidemiology Program, Perelman School of Medicine University of Pennsylvania
2002-2005	Co-Director, Master of Science in Clinical Epidemiology Program, Perelman School of Medicine University of Pennsylvania
2006-2012	Deputy Director, Clinical Epidemiology Unit, Perelman School of Medicine University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics
2011-present	Director, Center for Therapeutic Effectiveness Research, Perelman School of Medicine University of Pennsylvania
2012-2013	Acting Director of the Division of Clinical Epidemiology and the Clinical Epidemiology Unit, Perelman School of Medicine University of Pennsylvania
2013-present	Director of the Division of Clinical Epidemiology and the Clinical Epidemiology Unit, Perelman School of Medicine University of Pennsylvania

Other Appointments:

1994-present	Senior Scholar, Perelman School of Medicine University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics (CCEB)
1997-present	Member, Graduate Group in Epidemiology and Biostatistics, Biomedical Graduate Studies Program, Perelman School of Medicine University of Pennsylvania
2003-present	Senior Fellow, Leonard Davis Institute
2011-present	Senior Fellow, Center for Behavioral Health Research, University of Pennsylvania

Specialty Certification:

1991	Board Certified in Internal Medicine (recertified in 2001)
1996	Board Certified in Cardiology (recertified in 2001)
2001	American College of Epidemiology

Licensure:

1991-Present	Commonwealth of Pennsylvania

Awards, Honors and Membership in Honorary Societies:

1984	Phi Beta Kappa
1984	Sigma Xi Research Society
1984	Sigma Xi Award for Outstanding Research in Chemistry
1988	Alpha Omega Alpha
1993	American College of Cardiology/Merck Adult Cardiology Fellowship Award
1996	Award for Best Poster, International Conference on Pharmacoepidemiology
2001	Excellence in Teaching in Epidemiology Award, Master of Science in Clinical Epidemiology Program
2001	Leonard Berwick Memorial Teaching Award
2003	ISPE Pharmacoepidemiology and Drug Safety Best Paper Prize
2004	UPHS Quality and Safety Award for Controlling Hypertension at Penn (CHAP)
2007-2008	Excellence in Teaching Epidemiology Award
2009	Honorable Mention, ISPE Pharmacoepidemiology and Drug Safety Best Article Award - "Moderate and high affinity serotonin reuptake inhibitors increase the risk of upper gastrointestinal toxicity"
2010-2011	Lindback Award for Distinguished Teaching
2014	Clinical Research Forum Top 10 Clinical Research Achievements for 2014

Memberships in Professional and Scientific Societies and Other Professional Activities:

International:

1994-Present	International Society of Pharmacoepidemiology (Member 1994-2004, Fellow 2004-present)
1998-2001	ISPE Educational Committee (Member)
2000-2001	ISPE Board of Directors (Member)

National:

1984-Present	Sigma Xi Research Society (Member)
1996	American College of Cardiology (Member, Subcommittee to Write Statement on "Development & Maintenance of Competence in Coronary Intervention Procedures")
1996-Present	American College of Cardiology (Fellow)
1996-Present	American Heart Association Council on Epidemiology and Prevention (Member)
1996-Present	American Heart Association and American Heart Association Council on Epidemiology and Prevention (Fellow)

1996-Present	Society for Cardiac Angiography and Interventions (Elected Consultant)
1998-1999	American College of Cardiology Scientific Sessions (Chair of Session)
1999-Present	Society for Epidemiologic Research (SER) (Member)
2000-Present	American Society for Clinical Pharmacology and Therapeutics (ASCPT) (Member)
2000-2003	Program Committee, National American Heart Association Council on Epidemiology (Member)
2001	Advisory Group, "One of a Kind", American Heart Association Quality of Care and Outcomes Research Expert Panel (Member)
2001-Present	American College of Epidemiology (Member)
2001-2002	National Peer Review Committee, American Heart Association Outcomes Research (Member)
2001-2002	Quality of Care and Outcomes Research Network of Experts, American Health Association (Appointed Member)
2002	Advocacy Committee, Epidemiology and Prevention Council of the American Heart Association (Member)
2002-2004	American College of Cardiology, National Cardiovascular Data Registry, Scientific and Clinical Support Task Force (Member)
2002-Present	National Institutes of Health, National Heart, Lung, and Blood Study Section (Ad Hoc Member reviewing all K-grants)
2002	Session at the Genomics Revolution: Bench to Bedside to Community and the 42nd Annual Conference on Cardiovascular Disease and Epidemiology and Prevention (Chair)
2006-Present	American College of Epidemiology (Fellow)
2007-Present	American Society for Clinical Investigation (ASCI) (Member)
2008-Present	American Society of Human Genetics (Member)
2008-Present	Pharmacogenetics Research Network (PGRN) (Affiliate Membership)
2008-Present	Member, Adult Congenital Heart Association

Local:

2000-2002 Health Measurement Task Force, Pennsylvania Delaware, American Heart Association (Member)

Editorial Positions:

1994-Present	Editorial Consultant, Journal of General Internal Medicine
1995-Present	Editorial Consultant, Annals of Internal Medicine
1996-Present	Editorial Consultant, The Journal of the American Medical Association
1997-Present	Editorial Consultant, Journal of the American College of Cardiology
1997-Present	Editorial Consultant, American Heart Journal
1997-Present	Editorial Consultant, New England Journal of Medicine
1999-Present	Associate Editor for North America, Pharmacoepidemiology and Drug Safety
2002-Present	Editorial Consultant, American Journal of Cardiovascular Drugs
2002-Present	Editorial Consultant, American Journal of Cardiology
2002-Present	Editorial Consultant, Circulation
2002-Present	Editorial Consultant Archives of General Psychiatry
2008-Present	Editorial Consultant, Blood

Academic and Institutional Committees:

1997-2005	Member, Master of Science in Clinical Epidemiology Admission Committee
1997-Present	Member, CCEB Graduate Teaching Curriculum Committee
1998-2002	Member, BGS Curriculum/Academic Standards Committee
1998-2005	Member, PhD in Epidemiology Admission Committee
1998-2002	Chair, CCEB Awards Committee
1999-2001	Chair, CCEB Institutional Review Board Committee
2002-2005	Chair, Master of Science in Clinical Epidemiology Curriculum Committee
2003-Present	Member, Committee on Appointments and Promotions (COAP), Department of Biostatistics and Epidemiology, University of Pennsylvania School of Medicine
2007-Present	Chair, Division of Epidemiology Recruitment Committee
2007-2008	Chair, University of Pennsylvania Bioethics External Review Committee
2008-Present	Chair of the Think Health Scientific Advisory Committee

Major Academic and Clinical Teaching Responsibilities:

1994-1996	Medicine 100 for Medical Students, Fall Semester (Penn Medicine)
1994-1998	Medicine 101A for Medical Students, Spring Semester (Penn Medicine)
1994-2000	Preceptor for EP 154, Medical Student Epidemiology Course, Spring/Fall Semester (Penn Medicine)
1994-Present	Attending Rounds at HUP and VA Medical Center (2 months/year)(Penn Medicine)
1994-Present	Clinic Supervisor, VA Medical Center and HUP (Penn Medicine)

1994-Present	Methods in Clinical Research. Seminar Series for Cardiology Fellows, Medical Residents, and Medical Students (Penn Medicine)
1994-Present	Faculty Preceptor, Cardiology Journal Club (Penn Medicine)
1998-Present	EP644 Cardiopulmonary Epidemiology, Advanced Master of Science in Clinical Epidemiology Course: Course Director and Teacher, Summer Semester (Penn Medicine)
2002-2005	Co-Director, MSCE Program at the University of Pennsylvania School of Medicine (30-40 Masters Students/year)
2006	"Adherence from the Academic Perspective. Setting a Research and Action Agenda to Increase Healthy Behaviors and Adherence.", Leonard Davis Institute, University of Pennsylvania, Philadelphia, PA
2008	"Warfarin Pharmacogenetics", The Children's Hospital of Philadelphia Pediatric Care Research Lecture, Philadelphia, PA

Lectures by Invitation:

Oct, 1995	"Calcium Channel Blockers: What Should We Do in the Meantime?"-Medicine Grand Rounds, Montgomery Hospital, Norristown, PA
May, 1996	"Current Controversy with Calcium Channel Blocker Use: What do we do now? Lehigh Valley Hospital Regional Symposium Series, Sixteenth Annual Update in Cardiology, Lehigh Valley Hospital, Allentown, PA
Jul, 1996	"Calcium Channel Blockers: What Should We Do in the Meantime?" - Medical Grand Rounds, Hospital of the University of Pennsylvania, Philadelphia, PA
Oct, 1996	"Calcium Channel Blockers: What Should We Do in the Meantime?" - Medical Grand Rounds, Chestnut Hill Hospital, Philadelphia, PA
Dec, 1996	"Current Controversy Over Calcium Channel Blockers" - Mid-Atlantic Chapter of the American College of Clinical Pharmacy, Philadelphia, PA
Jan, 1997	"Aspirin and 'Primary' Prevention of Cardiovascular Disease" - US Food and Drug Administration, Gaithersburg, MD
May, 1998	"Drug Safety Case Reports: From Calcium Blockers to FenPhen" - American Society of Hypertension, 13th Annual Meeting, New York, NY
Jun, 1999	"Sexual Activity and Cardiac Risk - Epidemiology" - International Consensus Conference - Sexual Activity and Cardiac Risk, Princeton, NJ
Dec, 1999	"The Health Risks of Obesity" - 1999 AHPA Ephedra International Symposium, Arlington, VA
Dec, 1999	"Coronary Stents", Cardiology Grand Rounds, Hospital of the University of Pennsylvania", Philadelphia, PA

Mar, 2000	"Clinical Epidemiology-What Qualifies As A Valid Study and What Isn't Ready For Prime Time?" ACC Medical Writers Symposium (in conjunction with the American College of Cardiology 49th Annual Scientific Session), Anaheim, CA
Aug, 2000	"Review of Available Data on Ephedra Alkaloids" Department of Health and Human Services Office of Women's Health, Washington, DC
Sep, 2000	"Coronary Stents: The Good, the Bad, or the Ugly?" Northwestern University School, Cardiology Grand Rounds, Chicago, IL
Mar, 2001	"Common Sense and Statistics in Identifying the High-Risk Patient" American College of Cardiology 50th Annual Scientific Session (ACC 2001), Orlando, FL
Mar, 2001	"Clinical Outcomes Research in Cardiology: A Broad Range of Research Opportunities" 41st Annual Conference on Cardiovascular Disease Epidemiology and Prevention, San Antonio, TX
Nov, 2001	"Volume-Outcome Relationship for PCI and CABG: Lessons from Registries" 2001 American Heart Association Scientific Sessions, Anaheim, CA
Nov, 2001	"Non-Steroidal Anti-Inflammatory Medications and Myocardial Infarction: Study Designs Issues" Epidemiology Advisory Panel, Pfizer, New York, NY
Jan, 2002	"The Epidemiology of Antidepressant Therapy" Duke University Depression and Cardiovascular Disease Meeting, Baltimore, MD
Mar, 2002	"Assisting Trained Clinicians to Become Researchers" 60th Association of Teachers of Preventive Medicine Annual Meeting, Washington, DC
Jun, 2002	"Volume and Outcomes in Primary Angioplasty for Acute MI." Ohio-American College of Cardiology Annual Meeting, Huron, OH.
Jun, 2002	"NSAIDs and COX-2 Inhibitors: Good for You, or Dangerous?" Hospital of the University of Pennsylvania, Cardiology Grand Rounds, Philadelphia, PA.
Oct, 2002	"NSAIDs, Aspirin and COX-2 Inhibitors: Risky Business or Unexpected Benefits?" University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics Seminar, Philadelphia, PA
Nov, 2002	"Selection of Pharmacological Approaches for Patients with Cardiovascular Disease and Depression." 2002 American Heart Association Scientific Sessions, Chicago, IL.
Apr, 2003	"NSAIDs, COX-2 Inhibitors, and Cardiovascular Disease: Balancing the Potential Benefits and Risks." Stanford University, Stanford, CA
Nov, 2003	"SSRIs and Cardiovascular Disease." University of Pennsylvania Symposium. Psychiatry in Medicine: Medicine in Psychiatry, Philadelphia, PA
Sep, 2004	"Improving Anticoagulation Care." The Agency for Healthcare Research and Quality (AHRQ) Third Annual Patient Safety Research Conference, Arlington, VA

Nov, 2004	"Molecular Pharmacoepidemiology", GlaxoSmithKline Seminar, Collegeville, PA
Jun, 2005	"Genetic and Molecular Pharmacoepidemiology", DIA 41st Annual Meeting, Washington, DC
Feb, 2006	"Human Pharmacogenomic Epidemiology", National Institutes of Health, Interdisciplinary Research Centers Workshop, Bethesda, MD
Sep, 2006	"Coxibs NSAIDs and the Heart: Did We Get It Wrong?" Controversies and Conundrums in Cardiovascular Medicine 2006, 23rd Annual Santa Fe Colloquium on Cardiovascular Therapy, Santa Fe, NM
Dec, 2007	"Warfarin Pharmacogenomics: Ready for prime Time?" University of Connecticut Cardiology Grand Rounds, Farmington, CT
Mar, 2008	"Warfarin Pharmacogenomics: The FDA and the Science" Cardiology Grand Rounds, University of Colorado, Denver, CO
May, 2008	"Measured vs. Assumed Drug Dosing Histories in the Management of Oral Anticoagulation: Doses & INR Levels" Drug Information Association (DIA), Washington, DC
May, 2008	"Methods and Approaches to Clinical Research in Adult Congenital Heart Disease" Research Symposium, 2008 Fifth National ACHA Conference, Philadelphia, PA
Sep, 2008	"Key research and policy issues to improve outcomes of anticoagulation therapies" AHRQ Centers for Education and Research on Therapeutics 7th Annual Partnerships to Advance Therapeutics Meeting, Bethesda, MD
Oct, 2008	"Warfarin Pharmacogenetics: Challenges and Opportunities" Pharmacogenetics Research Network (PGRN) Scientific and Steering Committee Meeting, University of North Carolina, Chapel Hill, NC
Nov, 2008	"Perspective on Genotype Guiding of Warfarin", Critical Path Institute's Warfarin Summit II, Bethesda, MD
Aug, 2009	"Warfarin", Introduction to Pharmacogenetic Epidemiology, 25th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Rhode Island Convention Center, Providence, RI
Aug, 2009	"Warfarin as a Prototype Pharmacogenetic Example", Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 25th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Rhode Island Convention Center, Providence, RI

Oct, 2009	"Design of the Clarification of Optimal Anticoagulation through Genetics (COAG) Trial", Inaugural Meeting of the Genomic Applications in Practice and Prevention Network (GAPPNet), sponsored by the CDC Office of Public Health Genomics, Ann Arbor, MI
Aug, 2010	"Warfarin as a Prototype Pharmacogenetic Example," Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 26th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Brighton, UK
Aug, 2010	"Warfarin Pharmacogenetics: A Clinical Trial Perspective", Duke Clinical Research Institute, Pharmacogenomics in Cardiovascular Disease: Balancing Scientific Promise with Clinical Reality, McLean, VA
Oct, 2010	"Adherence and Novel Methods", Duke Clinical Research Institute, Medication Adherence: What do we need to do to get people to take their medicines? McLean, VA
Oct, 2010	"Warfarin Pharmacoepidemiology", The Eighth International Workshop on Pharmacodynamics of Anticancer Agents, Hakone, Japan
Nov, 2010	"How Do We Bring Pharmacogenetics into Practice?", FIP Pharmaceutical Sciences 2010 World Congress, AAPS Annual Meeting and Exposition, New Orleans, LA
Nov, 2010	"Effectiveness and Safety Research: Translation Into Practice", Kaiser Permanente, San Francisco, CA
Apr, 2011	"New incentive approaches for adherence." University of Pennsylvania, Emerging Statistical Issues in the Conduct and Monitoring of Clinical Trials, University of Pennsylvania Annual Conference on Statistical Issues in Clinical Trials, Philadelphia, PA
Aug, 2011	"Warfarin as a Prototype Pharmacogenetic Example," Introduction to Pharmacogenetic Epidemiology, Keynote Speaker, 27th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Chicago, IL
Apr, 2012	"Personalized Medicine: The Promise and the State of the Science" University of the Sciences, Making the Connections: Personalized Medicine - From Promise to Public Health and Policy Symposium and panel discussion, Philadelphia, PA
Apr, 2012	"Therapeutic Effectiveness Research or Why "We Can't Solve Problems By Using the Same Kind of Thinking We Used When We Created Them."" Hospital of the University of Pennsylvania Cardiology Grand Rounds, Philadelphia, PA
Aug, 2012	"Pharmacogenetics: Clinical Utility Studies and Other True-Life Examples" Introduction to Pharmacogenetic Epidemiology, 28th International Conference on Pharmacoepidemiology and Therapeutic Risk Management, Barcelona, Spain

Apr, 2013	"Personalized Medicine in Cardiology", Utrecht University Symposium - The future of Anticoagulation Therapy, Amsterdam, Netherlands
Apr, 2013	"Why We Are Failing to Deliver Optimal Medical Care: A Case for Therapeutic Effectiveness Research" Northwestern University Grand Rounds, Chicago, IL
May, 2013	"Bringing Pharmacogenetics into Practice", Children's Hospital of Philadelphia Pediatric Cardiology Research Lecture, Philadelphia, PA
Aug, 2013	"Pharmacogenetics: Clinical Utility and Clinical Adoption", 29th International Conference on Pharmacoepidemiology & Therapeutic Risk Management, Montreal, Canada
Nov, 2013	"The Clarification of Optimal Anticoagulation through Genetics (COAG) Trial", American Heart Association Scientific Sessions, Dallas, TX

Organizing Roles in Scientific Meetings:

2001	Member, Program Committee, National American Heart Association Council on Epidemiology New Orleans, LA
2002	Member, Program Committee, National American Heart Association Council on Epidemiology Honolulu, HI
2003	Member, Program Committee, National American Heart Association Council on Epidemiology Miami, FL
2006	ISPE, International Conference on Pharmacoepidemiology, Committee on Pharmacogenomics Lisbon, Portugal
2009	ISPE Special Interest Group in Pharmacogenomics Providence, RI
Nov, 2010	Session Chair, "Pharmacogenetics: Found in Translation?", FIP Pharmaceutical Sciences 2010 World Congress, AAPS Annual Meeting and Exposition New Orleans, LA
2010	"Medication Adherence: Specific Recommendations to Transform Clinical Care" Session II Co-Chair, Duke Clinical Research Institute, Medication Adherence: What do we need to do to get people to take their medicines? McLean, VA

Grants:

Pending:

Dynamic Prediction Modeling for Generalizable Risk, NIH, 8/2015-7/2020 (French B/Kimmel S, PI), $524,041/annual direct costs, 30% effort (Role in grant: Co-PI, The likelihood a pt./population will develop a disease, respond to therapy, or have a clinical outcome is important for delivering high-quality pt. care/improving public health. Although methods have been developed to accomplish these goals, these models typically do not work in broad practice. We will develop a novel approach to ensure that risk-prediction models are useful/accurate when used in practice, enhance the delivery of care, and improve the public's health.)

New Quantitative Methods for Prediction of Re-hospitalization in Heart Failure, NIH, R01-HL122487, 4/2015-3/2019 (French B., PI), $175,000/annual direct costs, 10% effort (Role in grant: Co-Investigator, Patients with chronic diseases requiring hospitalization have difficulty managing their disease and are often readmitted to the hospital. Several risk-prediction algorithms have been derived to predict the occurrence of a CHF adverse event for which statistical analyses have been based on receiver operating characteristic curves/risk reclassification. Goal is to maximize the use of information collected on patients to inform more accurate predictions of future clinical status.)

Comparison of the Risk of Congestive Heart Failure Between Patients with Type 2 Diabets Mellitus Initiating Saxagliptin and Those Initiating Other Oral Antidiabetic Treatments, Astra Zeneca, N/A, 9/2014-8/2016 (LoRe V., PI), $427,625/annual direct costs, 2.5% effort (Role in grant: Co-Investigator, To compare the incidence of hospitalized CHF events among patients with T2DM who are new initiators of saxagliptin and those who are new initiators of OADs in classes other than DPP-4 inhibitors.)

Current:

Genomic Medicine Pilot Demonstration Projects Coordinating Center, NIH/NIHGRI, U01-HG007266 , 6/2013-4/2017 (Kimmel SE, PI), $400,000/annual direct costs, 21.33% effort (Role in grant: PI, The goal of the Coordinating Center at the University of Pennsylvania is to ensure the success of individual Genomic Demonstration Pilot Projects while stimulating collaboration across projects to produce and disseminate generalizable knowledge that can be used to expand and sustain existing efforts and to promote implementation in other settings and for other diseases.)

Patient and Provider Perspectives on Reasons for Hospital Readmissions, Patient Centered Outcomes Research Institute (PCORI), IP2-P10000186, 10/2012-6/2015 (Kimmel, S., PI), $249,925/annual direct costs, 15% effort (Role in grant: PI, Patients with chronic illnesses that require hospitalization, such as heart failure, often have difficulty managing their diseases after discharge and are often readmitted to the hospital within a short period. Efforts to predict and reduce readmissions have not been tailored to the specific needs of patients most at risks for readmission.)

Enabling Medical Research Growth in Emergency Medicine, NIH, K12-HL109009, 7/2011-6/2016 (Kimmel SE, PI: Becker L, Co-Investigator), $161,750/annual direct costs, 3.75% effort (Role in grant: Co PI, This proposed program will emphasize research training in diagnosing and managing patients with acute, life threatening emergencies, especially life threatening cardiovascular diseases, to address the major national shortage of qualified clinical researchers in emergency care research.

Clinical Outcomes of Saxagliptin, AstraZeneca and Bristol-Myers Squibb, 8/2010-8/2017 (Strom, B., PI: Kimmel, S., Co-Investigator), $1,032,225/annual direct costs, 5% effort (Role in grant: Co-Investigator, This project will provide information on utilization and outcomes of saxagliptin.)

Comparative Effectiveness Of Alternative Levels Of Stroke, Agency For Healthcare Research And Quality (Ahrq), 5-R01-HS-018540-04-REVISE, 9/2009-7/2015 (Kimmel SE, PI), $325,399/annual direct costs, 5% effort (Role in grant: PI, This research is expected to greatly expand the limited body of empirical knowledge of relevance of acute rehabilitation following stroke. Such evidence-based knowledge will be essential to guide future practice and policies in the VHA and private sectors alike.)

Do Amputees Benefit From Comprehensive Rehabilitation Services, National Institute Of Child Health And Human Development/Nih/Dhhs, 5-R01-HD-042588-08 REVISE, 6/2009-5/2015 (Kimmel SE, PI), $228,236/annual direct costs, 5% effort (Role in grant: PI, This ongoing study of veteran amputee population represents a first attempt to image the full continuum of acute rehabilitation, and long-term care services received by a defined group of patients. It further includes various types of long-term 3 year outcomes. We anticipate that this research will greatly expand the limited body of empirical knowledge of relevance to the rehabilitation and post-acute care of amputees.)

Clinical Importance of Drug-Drug-Interactions, NIH, R01-AG025152, 12/2004-3/2017 (Hennessy, S., PI: Kimmel, S., Co-Investigator), $317,381/annual direct costs, 1.91% effort (Role in grant: Co-Investigator, Propose to extend our approach to population DDI studies in 2 ways: 1) by incorporating mechanistic considerations, increasing translation to/from the "left hand" side of the translational spectrum; 2) implementing a sequential approach of identifying/quantifying risk, then, based on this step, characterizing important DDIs, with the goal of providing clinical detail needed for future efforts to mitigate risk;)

Continuation of the Chronic Renal Insufficiency Cohort (CRIC) Study, NIH-NIDDK, U01-DK060990, 9/2001-4/2018 (Feldman, H., PI), $1,634,607/annual direct costs, 2% effort (Role in grant: Co-Investigator, The objective of this study is to focus on the prediction and mechanisms of progressive renal disease and cardiovascular events in patients with CRI.)

Past:

Development of Novel Methods for Comparative Effectiveness Clinical Trials, NCRR, UL1-RR024134 , 8/2011-6/2013 (Kimmel, S., PI), $183,060/annual direct costs, 15% effort (Role in grant: PI, Comparative Effectiveness research (CER) of therapeutic strategies must often serve a dual role: Be rigorous yet also relevant to clinical practice. Often, a decision is made to sacrifice one for the other. Although clinical trials are ideal for producing unbiased comparative effectiveness results, they typically study a single intervention at a single point in time and thus do not inform the way that many existing therapies are used in actual clinical practice.)

Adherence to Antidepressant Medication and Hypertension Treatment, NIH, R34, 12/2009-7/2013 (Bogner, H., PI), $148,936/annual direct costs, 1% effort (Role in grant: Co-Investigator, The primary aims of this proposal are to design & test an integrated intervention strategy.)

Data Infrastructure for Post-marketing Comparative Effective, NIH, RCI-AG025152, 9/2009-8/2012 (Hennessy, S., PI), $332,891/annual direct costs, 1% effort (Role in grant: Co-Investigator, To demonstrate the utility of this data resource for comparative effectiveness research by examining the comparative effectiveness of different strategies for initial supplementation and monitoring of potassium (K) in patients newly starting loop or thiazide diuretics with regard to preventing a) all-cause death and b) sudden death/ventricular arrhythmia.)

Developing Interactive Technologies to Improve Research and Health Behavior , NIH, RC2-AG036592, 9/2009-9/2011 (Asch, D., PI: Volpp, K., Co-Investigator), $1,757,058/annual direct costs, 2% effort (Role in grant: Co-Investigator)

The Risk of Myocardial Infarction in Patients with Psoriasis, NIH, R01-HL099744, 3/2009-1/2015 (Gelfand, J., PI), $499,195/annual direct costs, 10% effort (Role in grant: Co-Investigator, The goal of this project is to determine the risk of MI and stroke in patients with psoriasis.)

A Randomized Trial of Interventions to Improve Warfarin Adherence, NIH, R01-HL090929, 9/2008-5/2012 (Kimmel, S., PI), $499,312/annual direct costs, 1.5% effort (Role in grant: PI, A randomized trial to evaluate the effectiveness and cost-effectiveness of two novel approaches aim to improve anticoagulation control by increasing warfarin adherence.)

Genetic and Environmental Determinants of Warfarin Response, Sub to UAB/NIH, R01 HL092173, 5/2008-4/2014 (Limdi, N., PI: Kimmel, S., Co-Investigator), $44,468/annual direct costs, 9% effort (Role in grant: Co-PI, The primary objective of the study is to quantify the association between warfarin dose requirements and genetic variants to develop and validate dosing algorithms.)

Randomized Trial of Genotype-Guided Dosing of Warfarin Therapy, NIH, N01, 4/2008-4/2014 (Kimmel, S., PI), $1,674,325/annual direct costs, 30% effort (Role in grant: PI, The primary aim of this program project grant is to identify predictors of adherence with warfarin therapy and develop a prediction rule to identify patients at risk for poor adherence with warfarin.)

Cardiovascular Safety of Stimulant Medications for Attention Deficit Hyperactivity Disorder, Shire, N/A, 3/2008-6/2011 (Hennessy, S., PI), $540,789/annual direct costs, 2.5% effort (Role in grant: Co-Investigator)

Lottery Based Approach to Improving Warfarin Adherence, Penn/CMA Aetna, 9/2007-8/2009 (Kimmel, S., PI), $398,994/annual direct costs, 1% effort (Role in grant: Co-PI)

Clinical Risk Factors for Primary Graft Dysfunction, NIH, R01-HL087115, 8/2007-7/2012 (Christie, J., PI), $632,632/annual direct costs, 2.5% effort (Role in grant: Collaborator, We aim to study the association of candidate genes in donors and recipients with subsequent development of PGD.)

Collaboration to Reduce Disparities in Hypertension, Pfizer, 3/2007-2/2009 (Kimmel, S., PI), $667,677/annual direct costs, 1% effort (Role in grant: PI)

Genetics of Primary Graft Dysfunction, NIH, R01-HL081619, 2/2007-1/2012 (Christie, J., PI), $574,259/annual direct costs, 2.5% effort (Role in grant: Co-Investigator, We aim to study the association of candidate genes in donors and recipients with subsequent development of PGD.)

Incidence and Predictors of Abnormal Liver Associated Enzymes in Patients with Atrial Fibrillation in a Routine Clinical Care Population, GlaxoSmithKline, 9/2005-5/2006 (Lewis, J., PI), $151,642/annual direct costs, 10% effort (Role in grant: Co-PI)

Drug-Induced Sudden Death & Ventricular Arrthythmia, NIH, R01 HL076697, 1/2005-11/2009 (Hennessy, S., PI), $458,173/annual direct costs, 2.5% effort (Role in grant: Co-Investigator)

Transdisciplinary Research on Genetics of Complex Traits, NIH, 9/2004-7/2009 (Kimmel, S., PI), $380,039/annual direct costs, 1% effort (Role in grant: PI)

Evaluation of the Controlling Hypertension at Penn (CHAP) Pilot Project, NIH, 1/2004-12/2005 (Sean Hennessy, PharmD, PI), $138,028/annual direct costs, 2% effort (Role in grant: Co-Investigator)

Value of Electronic Clinical Records for Medical Research, NIH, 7/2003-6/2008 (Tannen, R., PI), $415,159/annual direct costs, 5% effort (Role in grant: Co-Investigator)

Collaboration to Reduce Disparities in Hypertension, PA Department of Health, 3/2003-2/2007 (Kimmel, S., PI: Volpp, K., Co-Investigator), $1,001,253/annual direct costs, 1% effort (Role in grant: PI)

A Prediction Rule for Post-Coronary Artery Bypass Grafting (CABG) Supraventricular Tachycardias, NIH, 12/2002-6/2007 (Rod Passman, MD, PI), $74,625/annual direct costs (Role in grant: Collaborator)

COX-2 Inhibitors and Cardiac Outcomes, Merk & Co, Inc., 11/2002-4/2003 (Stephen E. Kimmel, MD, MSCE, PI), $250,000/annual direct costs, 2% effort (Role in grant: PI)

Patient Oriented Research in Anticoagulation, NIH, 9/2002-8/2008 (Kimmel, S., PI), $123,894/annual direct costs, 50% effort (Role in grant: PI)

Selective Serotonin Reuptake Inhibitors and Cardiac Outcomes, Glaxo-SmithKline Beecham, 1/2002-1/2003 (PI, PI), $163,881/annual direct costs, 2% effort (Role in grant: PI)

Prospective Cohort Study of Chronic Renal Insufficiency, NIH, 9/2001-6/2008 (Feldman, H., PI), $790,753/annual direct costs, 3.5% effort (Role in grant: Co-Investigator)

Improving Patient Safety by Reducing Medication Errors, NIH, 9/2001-8/2006 (Strom, B., PI), $867,334/annual direct costs, 15% effort (Role in grant: Project Leader)

Heart Sense: A Game for Reducing Heart Attack Pre-Hospitalization Delay-Phase II, NIH, 9/2001-8/2003 (Silverman, PI), $54,115/annual direct costs, 8% effort (Role in grant: Co-Investigator)

Neurohormonal Activation in Pulmonary Hypertension, NIH, 7/2001-6/2006 (Kawut, S., PI), $130,180/annual direct costs (Role in grant: Co-Investigator)

Prediction of Warfarin Dosing Using Clinical and Genetic Factors., NIH, R01-HL066176, 3/2001-10/2014 (Kimmel, S., PI), $515,039/annual direct costs, 2% effort (Role in grant: PI, A prospective cohort study that will incorporate patient, environmental factors, and genetic variants to develop and validate dose prediction models.)

Predictors of Anticoagulation Control on Warfarin, NIH, 3/2001-1/2008 (Kimmel, S., PI), $502,197/annual direct costs, 30% effort (Role in grant: PI)

Clinical risk factors and biomarkers of oxidant stress in primary graft failure following lung transplantation, NIH, 7/2000-6/2005 (Jason Christie, MD, PI), $120,847/annual direct costs (Role in grant: Collaborator,)

Risk of Upper Gastrointestinal Bleeding with OTC NSAIDs, Bayer Consumer Care, 7/2000-12/2003 (Jim Lewis, MD, PI), $397,922/annual direct costs, 10% effort (Role in grant: Co-Investigator)

AIDS Clinical Trials Unit, NIH, 1/2000-12/2004 (Friedman, PI), $7,684,407/annual direct costs, 4% effort (Role in grant: Co-Investigator)

COX-2's and Myocardial Infarction, Searle Pharmaceutical, 1/2000-4/2003 (Stephen E. Kimmel, MD, MSCE, PI), $268,547/annual direct costs (Role in grant: PI)

Health Effects of Schizophrenia and Antipsychotic Medications, Pfizer, 12/1999-3/2001 (Sean Hennessy, PharmD, PI), $218,375/annual direct costs, 7.5% effort (Role in grant: Co-Investigator)

Protamine Mortality Study, Pharming, 11/1999-4/2001 (Stephen E. Kimmel, MD, MSCE, PI), $38,866/annual direct costs, 6% effort (Role in grant: PI)

Heart Sense: A Game for Heart Attack Symptom Training, NIH, 10/1999-9/2000 (Silverman, PI), $68,244/annual direct costs, 4% effort (Role in grant: Co-Investigator)

Early Determination of Stroke Subtype with CT Angiography, NIH, 7/1999-6/2004 (Scott Kasner, MD, PI), $132,891/annual direct costs (Role in grant: Collaborator)

Left Ventricular Geometry and Cardiac Risk in Blacks, NIH CAP Award, 7/1998-6/2003 (Martin Keane, MD, PI), $81,345/annual direct costs (Role in grant: Co-Investigator)

VA Stars and Stripes Healthcare Network Competitive Pilot Project, Veteran's Administration, 7/1998-6/2000 (Michael Berkwits, PI), $24,550/annual direct costs (Role in grant: Supervisor of PI)

Risk Factors for Valve Abnormalities Among Users of Fenfluramine or Dexfenfluramine: A Case-Series and Case Control Study, Wyeth-Ayerst, 2/1998-6/2003 (Stephen E. Kimmel, MD, MSCE, PI), $1,516,393/annual direct costs, 20% effort (Role in grant: PI,)

Prevention of Myocardial Infarction by Non-Steroidals, NIH, 9/1997-8/2002 (Stephen E. Kimmel, MD, MSCE, PI), $432,914/annual direct costs, 8% effort (Role in grant: PI)

Rates and Predictors of Repeat PTCA, COR Therapeutics, Inc., Key Pharmaceuticals, Inc., 5/1997-4/1999 (Stephen E. Kimmel, MD, MSCE, PI), $222,762/annual direct costs, 4% effort (Role in grant: PI)

Acute Myocardial Infarction and use of Nicotine Patches, Marion Merrell Dow, CIBA Pharmaceuticals & McNeil Labs, 12/1994-6/2000 (Stephen E. Kimmel, MD, MSCE, PI), $453,765/annual direct costs, 10% effort (Role in grant: PI)

Determining Incidence and Risk of Protamine Reactions, NIH, , 9/1994-8/1999 (Stephen E. Kimmel, MD, MSCE, PI), $82,000/annual direct costs, 80% effort (Role in grant: PI)

Protamine Reactions Following Cardiopulmonary Bypass, ACC/Merck Adult Cardiology Fellowship Award, 7/1993-6/1994 (Stephen E. Kimmel, MD, MSCE, PI), $35,000/annual direct costs, 80% effort (Role in grant: PI,)

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Research Publications, peer reviewed (print or other media):

1. Kimmel SE, Berlin JA, Laskey WK: The Relationship Between Coronary Angioplasty Procedure Volume and Major Complications. JAMA 274: 1137-1142, 1995.

2. Kimmel SE, Berlin JA, Strom BL, Laskey WK: Development and Validation of a Simplified Predictive Index for Major Complications in Contemporary Percutaneous Transluminal Coronary Angioplasty Practice. J Am Coll Cardiol 26: 931-938, 1995.

3. Strom BL, Berlin JA, Kinman JL, Spitz PW, Hennessy S, Feldman H, Kimmel S, Carson J: Parenteral Ketorolac and Risk of Gastrointestinal and Operative Site Bleeding. A Postmarketing Surveillance Study. JAMA 275: 376-382, 1996.

4. Feldman HI, Kinman JL, Berlin JA, Hennessy S, Kimmel SE, Farrar J, Carson JL, Strom BL: Parenteral Ketorolac: The Risk of Acute Renal Failure. Ann Intern Med 126: 193-199, 1997.

5. Hennessy S, Kinman JL, Berlin JA, Feldman HI, Carson JL, Kimmel SE, Farrar J, Harb G. Strom BL: Lack of hepatoxicity of parenteral ketorolac in the hospital setting. Arch Intern Med 157: 2510-2514, 1997.

6. Kimmel SE, Berlin JA, Hennessy S, Strom BL, Krone RJ, Laskey WK: Risk of Major Complications from Coronary Angioplasty Performed Immediately after Diagnostic Coronary Angiography: Results from the Registry of the Society for Cardiac Angiography and Interventions. J Am Coll Cardiol 30: 193-200, 1997.

7. Hirshfeld JW, Ellis SG, Faxon DP, Block PC, Carver JR, Douglas JS, Eigler NH, Hlatky MA, Holmes DR, Huter AM, Jacobs AK, Johnson WL, Jollis JG, Kimmel SE, Laskey WK, Luft H, Malenka DJ, Oboler AA, Summers AE, Taussig AS: ACC Clinical Competence Statement. Recommendations for the Assessment and Maintenance of Proficiency in Coronary Interventional Procedures. Statement of the American College of Cardiology. J Am Coll Cardiol 31: 722-743, 1998.

8. Kimmel SE, Sekeres MA, Berlin JA, Ellison N, DiSesa VJ, Strom BL: Risk Factors for Clinically Important Adverse Events after Protamine Administration following Cardiopulmonary Bypass. J Am Coll Cardiol 32: 1916-1922, 1998.

9. Kimmel SE, Sekeres MA, Berlin JA, Goldberg LR, Strom BL: Adverse Events after Protamine Administration in Patients Undergoing Cardiopulmonary Bypass: Incidence of Events and Risks and Predictors of Under-Reporting. J Clin Epidemiol 51: 1-10, 1998.

10. Berlin JA, Kimmel SE, Ten Have TR, Sammel MD: An Empirical Comparison of Several Clustered Data Approaches under Confounding due to Cluster Effects in the Analysis of Complications of Coronary Angioplasty. Biometrics 55: 470-476, 1999.

11. Conroy MB, Rodriguez SU, Kimmel SE, Kasner SE: Helicopter transfer offers a Potential Benefit to Patients with Acute Stroke. Stroke 30: 2580-2584, 1999.

12. Kimmel SE, Keane MG, Crary JL, Jones J, Kinman JL, Beare J, Sammel M, St. John Sutton M, Strom BL: Detailed Examination of Fenfluramine-Phentermine Users with Abnormalities Identified in Fargo, North Dakota. Am J Cardiol 84: 304-308, 1999.

13. Mahoney P, Kimmel SE, DeNofrio D, Wahl P, Loh E: Prognostic Significance of Atrial Fibrillation in Patients at a Tertiary Medical Center Referred for Heart Transplantation because of Severe Heart Failure. Am J Cardiol 83: 1544-1547, 1999.

14. Kasner SE, Kimmel SE: Accuracy of Initial Stroke Subtype Diagnosis-A Decision Analysis. Cerebrovasc Dis 10: 18-24, 2000.

15. Kimmel SE, Localio AR, Brensinger C, Miles C, Hirshfeld J, Haber HL, Strom BL: Effects of Coronary Stents on Cardiovascular Outcomes in Broad-Based Clinical Practice. Arch Intern Med 160: 2593-2599, 2000.

16. Krone RJ, Laskey WK, Johnson C, Kimmel SE, Klein LW, Weiner BH, Cosentino JJA, Johnson SA, Babb JD for the Registry Committee of the Society for Cardiac Angiography and Interventions: A Simplified Lesion Classification for Predicting Success and Complications of Coronary Angioplasty. Am J Cardiol 85: 1179-1184, 2000.

17. Laskey WK, Kimmel S, Krone RJ: Contemporary Trends in coronary Intervention: A Report From the Registry of the Society for Cardiac Angiography and Interventions. Catheterization and Cardiovascular Interventions 49: 19-22, 2000.

18. Mohler III ER, Klugherz B, Goldman R, Kimmel SE, Wade M, Sehgal CM: Trial of a Novel Prostacyclin Analogue, UT-15, in Patients with Severe Intermittent Claudication. Vasc Med 5: 231-237, 2000.

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20. Kimmel SE, Berlin JA, Miles C, Jaskowiak J, Carson JL, Strom BL: Risk of Acute First Myocardial Infarction and Use of Nicotine Patches. J Am Coll Cardiol 37: 1297-302, 2001.

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22. Passman R, Beshai J, Pavri B, Kimmel S: Predicting Post-Coronary Bypass Surgery Atrial Fibrillation from the Pre-Operative Electrocardiogram. Am Heart J 142(5): 806-10, 2001.

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29. Kimmel SE, Berlin J, Kinman J, Hennessy S, Feldman H, Carson J, Strom B: Parenteral Ketorolac and Risk of Myocardial Infarction. Pharmacoepidemiology and Drug Safety 11: 113-119, 2002.

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37. Christie JD, Kotloff RM, Pochettino A, Arcasoy SM, Rosengard BR, Landis JR, Kimmel SE: Clinical Risk Factors for Primary Graft Failure Following Lung Transplantation. Chest 124: 1232-1241, 2003.

38. Horwitz PA, Berlin JA, Sauer WH, Laskey WK, Krone RJ, Kimmel SE: Bleeding risk of platelet glycoprotein IIb/IIIa receptor antagonists in broad-based practice: results from the society for cardiac angiography and interventions registry. Am J Cardiol 91: 803-806, 2003.

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